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Trace Metals and other Contaminants in the Environment 10

Lead and Public Health

Science, Risk and Regulation

Paul Mushak



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Contents

Acknowledgments ix

Part 1

Lead in the Human Environment

1. Introduction 3
2. A Brief Early History of Lead as an Evolving
Global Pollutant and Toxicant 23
3. Lead in the Human Environment: Production,
Uses, Trends 41
4. Lead in the Human Environment: Lead Emissions
and Emission Trends 73
5. Lead in the Human Environment: Fate and
Transport Processes 91
6. Lead Concentrations in Environmental Media
Relevant to Human Lead Exposures 117

Part 2

Lead Exposure in Human Populations

7. Lead Exposure in Human Populations: Lead Intakes 217
8. Lead Exposure in Human Populations: Lead
Toxicokinetics and Biomarkers of Lead Exposure 243
9. Predictive Modeling Approaches for Assessing
Human Lead Exposure 317
10. The Environmental Epidemiology of Human Lead
Exposure 345

Part 3**Lead Toxicity in Humans**

- | | |
|---|-----|
| 11. Lead Toxicity in Humans: A Brief Historical Perspective and Public Health Context | 401 |
| 12. Neurotoxicity of Lead in Human Populations | 439 |
| 13. Cardiovascular Toxicity of Lead in Human Populations | 503 |
| 14. Reproductive and Developmental Toxicity of Lead in Human Populations | 537 |
| 15. The Nephrotoxicity of Lead in Human Populations | 567 |
| 16. Hematological Effects of Lead in Human Populations | 597 |
| 17. Carcinogenic and Genotoxic Effects of Lead in Human Populations | 635 |
| 18. Immunotoxic Effects of Lead in Human Populations | 671 |
| 19. Effects of Lead on Other Organs and Systems in Human Populations | 697 |

Part 4**Human Health Risk Assessment**

- | | |
|--|-----|
| 20. Human Health Risk Assessment for Lead: Introduction, Context, Rationale | 715 |
| 21. Hazard Characterization for Lead in Human Populations | 729 |
| 22. Dose–Response Relationships for Toxic Effects of Lead in Human Populations | 745 |
| 23. Exposure Characterizations for Lead in Specific Human Populations | 769 |
| 24. Health Risk Characterization of Lead Effects in Human Populations | 797 |

Historical Context	401
Populations	439
Human Populations	503
Toxicity of Lead in	537
Human Populations	567
Human Populations	597
Effects of Lead in	635
Human Populations	671
and Systems in	697
For Lead:	715
Lead in	729
For Toxic Effects	745
Lead in Specific	769
Lead Effects in	797

Part 5

Regulatory Approaches to Control

25. Legislative Aspects of Lead Regulation and Regulatory Policies	817
26. Lead Regulation and Regulatory Policies: Lead in Paint	841
27. Regulation and Regulatory Policies for Lead in Ambient Air	875
28. Regulation and Regulatory Policies for Lead in Water	899
29. Regulation and Regulatory Policies for Lead in Food	923
Index	945

Lead Concentrations in Environmental Media Relevant to Human Lead Exposures

6.1 LEAD AS MULTIMEDIA POLLUTANT

Figure 5.1 in the previous chapter presented a graphic depiction of lead as a substance which enters and departs multiple environmental compartments, multiple environmental media, with relative physical and chemical ease. This recognition and graphic characterization of lead as a multimedia pollutant is of recent vintage, dating to the 1977 U.S. EPA Air Quality Criteria for Lead report (U.S. EPA, 1977), the first of a series of Federal lead documents, and the 1980 report to the National Commission on Air Quality by Mushak and Schroeder (see also Chapter 25). Subsequent expert consensus treatises such as EPA's later Air Quality Criteria Documents for Lead (U.S. EPA, 1986a, 2006), the U.S. CDC Statements on Childhood Lead Poisoning (U.S. CDC, 1985, 1991, 2005), the NAS/NRC (1980, 1993), and the WHO (1995) helped establish the nature and extent of lead's multimedia behavior.

Lead's behavior as a multimedia pollutant poses problems for human health risk assessment and regulatory science at several levels, a number of which are presented in detail in later chapters. First, it is important to establish the full extent of lead exposures of human populations, especially those subsets of the population at elevated risk for exposure and/or harm. Establishing the extent of exposure mainly includes identifying and quantifying lead intakes and uptakes into the human body.

A second important factor for lead as a multimedia pollutant is the recognized toxicokinetic and toxicological fact that all sources of lead intake and uptake by human populations contribute to a single, integrated internal toxicological exposure or "dose." It is not biologically or toxicologically required that a single source of lead provides all of the potentially toxic lead exposure, merely that it provides a measurable contribution to the total. The body, furthermore, does not typically preserve the chemical form of the lead entering the receiving compartments, for example, the gastrointestinal and

the respiratory tracts. Once absorbed, a given quantity of lead is toxic to the same extent, regardless of its environmental chemical or physical form.

A third factor in assessing lead in multiple contact media and those associated lead exposures in human populations is that lead is biologically cumulative and is significantly cumulative over time in human populations. Lead is cumulative in humans principally because of its propensity to lodge in skeletal mineral tissue with time, growth, and age. The accumulation begins in childhood and continues well into adulthood (Mushak, 1993, 1998; U.S. EPA, 2006). However, lead is unlike methyl mercury, in that it does not biomagnify across species or trophic orders as one goes up the food web.

There are two consequences for risk assessment of this potential for accumulation in bone. First, lead lodged in bone can be mobilized under diverse physiological and other stresses, e.g., pregnancy, lactation, menopause, physical immobilization (Gulson et al., 1995, 1997; Markowitz and Weinberger, 1990; Silbergeld et al., 1988), and it subsequently enters the bloodstream, producing an "endogenous" pool of lead for inducing toxic effects. Secondly, lead's systemic accumulation places a premium on quantification of lead intakes and uptakes over much of the lifetime of human populations, since the main bone depository for lead, cortical bone, has a half-life for lead release of up to several decades from that compartment (Rabinowitz et al., 1976).

This chapter examines the levels of lead in environmental media with which human populations interact by such means as ingestion and inhalation. Quantification of lead levels in media not only applies to the ongoing and near-term case, but also requires a depiction of the environmental lead picture over population lifetimes. If lead deposited in bone 30 years ago can be released and produce harm, one needs to evaluate as best as possible the magnitude of lead intakes via various media 30 years ago. This would particularly be the case in attempting predictive modeling of lead exposures that occurred decades ago.

For measuring lead in environmental media providing potential human lead exposures, this chapter includes older published data for lead concentrations in media, data which are old enough to encompass the full lifetimes of living populations. This is because of long-term Pb storage in bone. One concern with any appraisal of older lead measurement data in media is that of analytical and statistical data reliability versus that of methods employed with more recent accepted techniques. Sensitivity is of particular concern. A potent toxicant such as environmental lead requires methods for quantification of concentrations of lead at ultra-trace levels in order to permit estimates of the full range of Pb exposures.

Evolution of methodologies for measuring lead in media and associated statistical analysis require a brief discussion of current methods in perspective. Methods have greatly improved over the years, so that one must confine reliance on older data to those likely to be most reliable.

ed, a given quantity of lead is toxic to the environmental chemical or physical form.

in multiple contact media and those associations is that lead is biologically cumulative over time in human populations. Lead is biologically cumulative because of its propensity to lodge in bone, and age. The accumulation begins in childhood (Mushak, 1993, 1998; U.S. EPA, 1995, 1997; Markowitz and Weinberger, 1995, 1997). Unlike methyl mercury, in that it does not follow trophic orders as one goes up the food web. Lead risk assessment of this potential for accumulation in bone can be mobilized under diverse circumstances, e.g., pregnancy, lactation, menopause, physical activity, 1995, 1997; Markowitz and Weinberger, 1995, 1997). Lead, once it subsequently enters the bloodstream, is a major concern for inducing toxic effects. Lead accumulation places a premium on quantification of lead in environmental media. Much of the lifetime of human populations, lead in bone, has a half-life for decades from that compartment (Rabinowitz

levels of lead in environmental media with such means as ingestion and inhalation. Lead in environmental media not only applies to the ongoing and past exposure, but also to the depiction of the environmental lead picture. Lead deposited in bone 30 years ago can be mobilized and needs to be evaluated as best as possible the lead in environmental media 30 years ago. This would particularly be true for predictive modeling of lead exposures that

environmental media providing potential human exposure. Lead in older published data for lead concentrations is not enough to encompass the full lifetimes of exposure. Lead in long-term Pb storage in bone. One conclusion from lead measurement data in media is that of the variability versus that of methods employed for measurement. Sensitivity is of particular concern. Lead in environmental media requires methods for quantification at ultra-trace levels in order to permit estimation of exposure.

For measuring lead in media and associated exposure, a discussion of current methods in perspective over the years, so that one must confine exposure to be most reliable.

6.2 SUMMARY OF SAMPLING AND LABORATORY ANALYTICAL METHODS FOR ENVIRONMENTAL LEAD

Environmental media of interest in this section on measurement are the same as those producing potential human lead exposures: ambient air, lead paints, diet, drinking water, soils and dusts, and some of the more problematic idiosyncratic sources. Sampling and laboratory measurement techniques now widely used are emphasized with comparative statements for older methods provided mainly to offer perspective. Biomarker sampling and measurement methodologies, i.e., procedures for lead in biological media directly relevant to human lead exposures, are presented in a later chapter.

Lead has been and continues to be so pervasive in human environments that it poses special challenges for sampling and analysis of either environmental or biological lead when present in extremely small amounts. Because of the contamination problem, no serious attempt at reliable lead measurements in environmental media can be done without rigorous quality assurance and quality control (QA/QC) protocols. For regulatory compliance with enforceable U.S. standards, such QA/QC steps are explicitly prescribed in order to have legal meaning (Code of Federal Regulations, CFR, 1982, 40:§58).

6.2.1 Analysis of Lead in Ambient Air

Sampling for ambient air lead measurements is quite complex in implementation and interpretation. Furthermore, that sampling complexity is arguably greater than it is for most other lead-containing environmental media. This arises partly from the nature of how human populations encounter lead in air and lead's fate and transport from points of emission. This section also summarizes some changes in sampling and methodology that have accompanied changes in lead emission sources. Specifically, lead from mobile sources and air lead analyses to quantify this source contribution have declined significantly in the last 15–20 years while stationary or point lead sources have increased in relative significance. The magnitude of these changes was noted earlier.

Methodologies for air lead sampling and laboratory analysis are limited to specific reference methods officially prescribed to accommodate the fact that air lead analysis in the United States is directed to, among other purposes, compliance with an ambient air lead standard within the framework of State Implementation Plans (SIPs). SIPs are the regulatory and legal means by which the various states implement the nationally enacted ambient air lead standard. The details are discussed in such treatises as Chapter 4 of the 1986 U.S. EPA Air Quality Criteria for Lead document (U.S. EPA, 1986a) and Chapters 2 and 3 of U.S. EPA, 2006. The required reference methods for legal enforcement are codified in the CFRs (CFR, 1982, 40:§58).

Three factors govern air lead sampling: (1) site selection; (2) appliances used for air sampling and form of lead being sampled; and (3) sample preservation prior to laboratory analysis. For any area's site sampling for airborne lead, some minimum number of sampling stations are required, depending on both population category and ranges of total suspended particulate (TSP). Air lead quantification reports, from earlier times through the present, typically reported air lead concentrations based on TSP. Other criteria pollutants have adopted size-selective sampling techniques with an eye to focusing on the most readily respirable particulate forms of the pollutants. These requirements mainly applied to those earlier years when the predominant contributor, on the order of 90–95% of total air lead, was leaded gasoline combustion.

Up to 6 to 8 air-monitoring stations were spelled out for areas with populations exceeding 500,000 and where prior testings showed particulate air levels within the "high" range, that is, when the TSP level exceeded 20% of the TSP standard. At the other extreme, areas with populations of only 50,000–100,000 and where the TSP level is less than the ambient air standard require no monitoring sites (Appendix D, CFR, 1982, 40:§58).

Ambient air lead partitions into vertical gradients, especially near mobile lead sources, e.g., vehicular exhaust from leaded gasoline combustion. There is special emphasis on those heights above source relevant to human lead exposures. Other specific monitor locating requirements include specifications for distances from roadways (5–100 m) and distances from obstacles between the monitor and the emitting source (Appendix E, CFR, 1982, 40:§58).

Ambient air sampling within typical regulatory and other contexts uses a high-volume ("hi-vol") aerosol sampler. Other collection devices may include filters, impactors, and impingers (U.S. EPA, 1971). This overall approach is based on the existence of lead in ambient air as largely inorganic particulate matter rather than in predominantly vapor form. Quantification using this sampler is as micrograms per cubic meter of air ($\mu\text{g Pb}/\text{m}^3$).

The dichotomous and impact samplers are other devices for air lead monitoring. The former collects particulate and segregates it into two size ranges: 0–2.5 μm and 2.5 to the maximum opening of the intake port, typically 10 μm (Loo et al., 1979). These ranges, generally conforming to fine and coarse particle categories, respectively, provide a close approximation to those particulate sizes that are relevant to respirable lead fractions and that fraction entering the deep pulmonary compartment.

Impactor samplers, which permit a broader range of measurable particulate size distributions, are constructed so that air entering at high flow rates encounters a series of trapping surfaces and particles are trapped based on size. First trapped are the coarsest particles followed sequentially by smaller and smaller fractions. The cascade samplers typically allow fractionations within narrow ranges, such as 0.01–0.1 μm (Dzubay et al., 1976).

sampling: (1) site selection; (2) appliances lead being sampled; and (3) sample preservers. For any area's site sampling for airborne sampling stations are required, depending on ranges of total suspended particulate (TSP). From earlier times through the present, techniques based on TSP. Other criteria pollutants sampling techniques with an eye to focusing on particulate forms of the pollutants. These requirements in earlier years when the predominant contributor of total air lead, was leaded gasoline

stations were spelled out for areas with populations where prior testings showed particulate air pollution. That is, when the TSP level exceeded 20% of the ambient level, areas with populations of only 10,000. TSP level is less than the ambient air standard. Appendix D, CFR, 1982, 40:§58).

to vertical gradients, especially near mobile sources. Just from leaded gasoline combustion. There are heights above source relevant to human lead exposure. For locating requirements include specifications (5–100 m) and distances from obstacles from emitting source (Appendix E, CFR, 1982,

typical regulatory and other contexts uses a high-volume sampler. Other collection devices may include impingers (U.S. EPA, 1971). This overall picture of lead in ambient air as largely inorganic and predominantly vapor form. Quantification is in micrograms per cubic meter of air ($\mu\text{g Pb}/\text{m}^3$). High-volume samplers are other devices for air lead monitoring. They filter and segregate it into two size ranges: greater than 10 μm opening of the intake port, typically in the 10–100 μm ranges, generally conforming to fine and coarse fractions. Collectively, provide a close approximation to the relevant to respirable lead fractions and that is the primary compartment.

to permit a broader range of measurable particulate matter. Constructed so that air entering at high flow rates through the surfaces and particles are trapped based on size. The largest particles followed sequentially by smaller particles. High-volume samplers typically allow fractionations of 1–0.1 μm (Dzubay et al., 1976).

Laboratory analysis of air lead contained on various sampler trapping materials typically uses reference methods, i.e., methods taken to be particularly reliable, that have relatively reliable track records and which have been codified for use to ascertain compliance with existing enforceable standards in the case of regulatory legal compliance testings (CFR, 1982, 40:§58).

The approved reference method for enforcement purposes (CFR, 1982, 40:§58) uses hi-vol samplers and measures lead by atomic absorption spectrometry (AAS). This laboratory method, which has been available in various analytical configurations for several decades, has been shown to be particularly reliable and sensitive for measuring lead quantitatively in a large range of environmental media. Flameless AAS is a more sensitive variation of this technique than conventional flame methods and has been the choice for many years. As with any lead measurement method, sample handling must minimize both contamination with lead and loss of lead from the sample. Comparatively, the contamination problem is still the more problematic and this is certainly the case for analyses in U.S. urban areas and in other industrialized nations (NAS/NRC, 1993; Patterson, 1983; Settle and Patterson, 1980). For air sample analyses, the codified reference method using AAS is quite adequate for a wide range of air lead concentrations.

Other methodologies fall into the category of definitive or alternative reference methodologies. The definitive method for lead, against which other methods are qualified for reference use, and the one employed for standard sample lead certification by the National Institute of Science and Technology (NIST), is isotope-dilution mass spectrometry (IDMS). Among its cardinal virtues, in addition to accuracy and precision, are sensitivity and applicability to many lead-containing environmental matrices.

6.2.2 Analysis of Lead in Paint

Lead in paint remains a major source of lead in human environments in terms of the U.S. national picture, and paint lead remains the dominant general lead source for humans residing in inner city, densely populated neighborhoods having a high fraction of deteriorated housing. Lead in paint, like lead in ambient air, is defined as a source of lead that works through pathways to provide human lead exposures. Shared pathways from these originating lead sources include interior and exterior dusts and yard soils. It is therefore appropriate to discuss this lead source with air lead and prior to discussions of lead analysis in pathway media such as soils, dusts, plants, water, and food. Paint lead also shares the characteristic of sampling complexity with air lead and, equally important, much of paint lead testing and quantification of lead in paint lies within regulatory and legal constraints that prescribe certain sampling and analysis protocols. Lead paint testings and assessments as part of risk assessment for U.S. housing and in other frameworks lie within the jurisdictions of two U.S. agencies. The principal agency

having oversight of lead paint-containing U.S. housing units is the U.S. Department of Housing and Urban Development (U.S. HUD, 1995), with the U.S. EPA sharing statutory mandated responsibility (U.S. EPA, 2001).

Sampling lead-painted surfaces is prescribed for the purpose of "... a surface-by-surface investigation to determine the presence of lead-based paint ...," the latter being present when any measured lead paint content in terms of lead loading is $\geq 1.0 \text{ mg/cm}^2$ or has a concentration $\geq 0.5\%$ lead by weight (CFR, 2001, 40: Part 745; CFR, 1996, 24: Part 35). Two types of residential units are covered in these regulations, single family units and multifamily units. In the latter case, statistical formulae are used to randomly select a fraction of all the units broken into two categories of multiunit age ranges, pre-1960 or 1960–1977. This is necessary since it usually would not be feasible to test the entire tally of units at, for example, an apartment complex. Table 7.3 of U.S. HUD (1995, Ch. 7) sets forth how many units are to be tested at a multiunit complex as a function of complex age.

Testings of lead-painted surfaces typically begin with *in situ* examination using portable X-ray fluorescence (XRF) spectrometers that have been calibrated and are of an acceptable type as prescribed by U.S. HUD regulations. There are statistical protocols that employ random sampling techniques that apply for either single residence or multifamily residences (U.S. HUD, 1995). Documenting surface samplings typically includes testing forms that spell out both the area of a residential unit tested, such as a living room, and components within that area, e.g., baseboards, window sills/sashes/wells.

A reading will produce one of three results: (1) a level below which a reading is considered negative; (2) an intermediate range where the result is deemed inconclusive; and (3) a reading which is positive, i.e., exceeds the inconclusive reading. Surfaces with inconclusive readings can be also examined by collecting paint chips at the same surface to determine whether the 0.5% lead chip standard has been exceeded.

Surfaces to be sampled are those often found to either be higher in paint lead content and/or within easy reach of those most likely to have exposure, i.e., infants and toddlers. They especially include window components—sills, frames/jambs, wells—easily accessible surfaces that have been commonly associated with lead exposures of very young children. Surfaces for testing besides painted areas are those that are varnished, stained, shellacked, or painted surfaces under wallpaper.

Frequency or other criteria for lead paint surface testing depends on the purpose of the effort. U.S. HUD (1995, Ch. 7, revised 1997) spells out two types of lead paint measurement protocols for paint surface testing. These are lead paint inspection testings and risk assessments, the latter to identify what HUD terms lead paint "hazards." The term "risk assessment" carries a regulatory definition (CFR 40: Part 745, 2001) focused on lead paint hazards. Lead paint inspections deal with determining the presence of lead paint and usually test many more areas than lead paint hazard risk assessments. Both

containing U.S. housing units is the U.S. Housing Development (U.S. HUD, 1995), with the stated responsibility (U.S. EPA, 2001).

is prescribed for the purpose of "... a to determine the presence of lead-based paint when any measured lead paint content in μcm^2 or has a concentration $\geq 0.5\%$ lead by (CFR, 1996, 24: Part 35). Two types of regulations, single family units and multi-family units, are used to randomly broken into two categories of multiunit age. This is necessary since it usually would not of units at, for example, an apartment complex (EPA, 1995, Ch. 7) sets forth how many units are to as a function of complex age.

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types of surface testing have gathering paint chips and how to collect the paint chips in their protocols, but the number of chips required is fewer with the risk assessment. This is because the risk assessment protocol is directed to chip sampling only in areas of deteriorated paint surfaces. In addition, as noted in more detail later, risk assessment of lead paint hazards entails lead paint dust testings and lead paint-impacted soil lead measurements.

There are other circumstances for which one or the other of the two types of surface testings is done. For example, collection of paint chip samples and laboratory testing are recommended for inaccessible or irregular surfaces that provide problems for XRF surface testings. *In situ* testing, on the other hand, is nondestructive and does not entail paint removal from portions of surfaces. Sampling for lead paint using chips, however, requires that the samples represent all layers of lead paint that could be present. Chips that are mainly surficial represent more recent, and usually lower lead or nonlead paint films. Surface delamination (broad surface peeling from poorly prepared surfaces), for example, produces chips that do not capture the earlier history of the entire paint depth on the residential surfaces. Such sampling would potentially result in understatement of likely lead exposure of child residents or visitors. The most common metric for paint chip lead measurement is percent by weight. To express lead content by area rather than percent by weight, a measured area of paint surface is typically obtained.

Portable XRF testing is the conventional method for *in situ* lead paint testings on older painted surfaces. The XRF method records lead present in all layers in the painted surface via full depth penetration of the X-rays. Irradiated lead from X- or gamma rays produces X-rays at a characteristic frequency or energy and at an intensity proportional to the integrated total amount of lead present throughout the irradiated layers. Legally prescribed performance by acceptable portable XRF analyzers is specifically spelled out by both the U.S. HUD and U.S. EPA with the inspector's use of an XRF Performance Characteristic Sheet. This form describes results in the context of analytical findings of negative, inconclusive, or positive readings. Where readings are less than either the threshold or the lower boundary of "inconclusive" the result is taken as negative, i.e., legally defined lead paint is absent. Similarly, the readings that exceed the upper boundary of the inconclusive range are taken as positive. The XRF Performance Characteristic Sheet also sets forth guidelines for calibration, substrate issues and corrections, etc.

Laboratory analysis of lead in paint chips should only be carried out by those laboratories certified for such analyses by the U.S. EPA National Lead Laboratory Accreditation Program (NLLAP), using approved laboratory methods. Use of AAS methodology, particularly in the ICP-AAS configuration, is the common approach. Another routine method is anodic stripping voltammetry (ASV).

The American Society for Testing Materials (ASTM) has provided validated procedures for quantification of lead in various types of paint samples. ASTM E 1645 prescribes preparation of paint lead chips for analysis while methods ASTM E 1613, ASTM E 1775, and ASTM PS 88 are directed to measurements of lead. The NIST has made paint standard reference materials (SRM) available for calibration and validation of lead paint measurement methodologies. SRM 2579 is available as a paint film while SRM 2589 consists of paint samples collected from interiors of various homes and prepared as a homogeneous powder, ~100,000 ppm (10%) lead, with >99% of the particles being <100 μm in size.

6.2.3 Analysis of Lead in Soils

The variety of sampling approaches for soil lead analyses is as complex as for lead paint. The type of sample taking for soils depends on the purpose of the analyses. Residential, industrial, and public area soils are often analyzed for regulatory, legal, risk assessment, or scientific research purposes. Soils may also be tested to quantify the amounts of lead present, to determine the source(s) of lead in the soils, and, in certain cases, to ascertain the chemical and physicochemical species of lead in the soils to ascertain both likely source and relative bioavailability of lead forms present.

A number of methodological and statistical criteria govern the actual physical nature of the soil sampling. There is the matter of spatial sampling, where one or more surface area soil samples are collected. Collection may entail grab sampling, where one sample is collected, or composite sampling, where subsamples are gathered and combined prior to analysis. Subsamples may also be analyzed individually before combining to provide a composite value. Compositing is typically done where there is some information available about the likely nature of the lead source providing the contamination and its distribution. That is, is the soil lead relatively uniform in distribution or heterogeneous, with one or more potentially troublesome "hot spots" that can produce human lead exposures (U.S. EPA, 1989; U.S. EPA 2008a, Child-Specific Exposure Factors Handbook)?

Soil sampling may also entail spatially variable collection in area or vertical depth. For example, if one is interested in whether exterior lead paint has weathered and released to adjacent soils, one would collect foundation perimeter, i.e., "drip-line" samples. If one is interested in whether roadways are affecting property front soils, samples can be collected at the curb. A critical factor in soil sampling is depth of sampling. Soil lead arising from atmospheric dry and wet deposition will typically deposit on the top 1–2 cm of soil surface. Absent other lead sources, deeper soil strata will have little lead content. Sample cores which are gathered well below this 1–2 cm depth have the potential to statistically dilute the soil lead loading with reference to lead exposure of young children who play in such soils and only come in

esting Materials (ASTM) has provided validation of lead in various types of paint samples. Preparation of paint lead chips for analysis while ASTM E 1775, and ASTM PS 88 are directed to ASTM. ASTM has made paint standard reference materials and validation of lead paint measurement available as a paint film while SRM 2589 from interiors of various homes and prepared SRM 2589 containing 100,000 ppm (10%) lead, with >99% of the

Soils

Methods for soil lead analyses is as complex as the sampling for soils depends on the purpose of sampling, and public area soils are often analyzed for environmental, or scientific research purposes. Soils are analyzed to determine the amounts of lead present, to determine the distribution of lead in the soils to ascertain both likely exposure and the variety of lead forms present.

Statistical and statistical criteria govern the actual sampling. There is the matter of spatial sampling, how many soil samples are collected. Collection may be by single sample is collected, or composite sampling, or by multiple samples and combined prior to analysis. Subsamples are collected before combining to provide a composite sample. This is done where there is some information available about the lead source providing the contamination. If the soil lead is relatively uniform in distribution, sampling is more potentially troublesome "hot spots" that are not uniform (U.S. EPA, 1989; U.S. EPA 2008a, 2008b; Handbook)?

For spatially variable collection in area or vertical, if one is interested in whether exterior lead paint is present on adjacent soils, one would collect foundation samples. If one is interested in whether roadways are a source, samples can be collected at the curb. The depth of sampling. Soil lead arising from roadways will typically deposit on the top 1–2 cm of soil. From other sources, deeper soil strata will have little lead. Samples are gathered well below this 1–2 cm depth. This dilute the soil lead loading with reference to the exposure of children who play in such soils and only come in

contact with the surface soil through typical child hand–mouth activity. Agricultural soils may require greater sampling depth or include deeper cores in multidepth sampling if one wishes to ascertain the subhorizon lead concentrations in crop root zones. Such deeper samplings, in tandem with surface testing, may also be required if Pb-impacted soils are tilled and turned over.

A critical factor in soil sampling is the question of lead content as a function of soil particle size distribution. Anthropogenic lead contamination of soils often entails deposition of lead particulate or suspended lead from runoff entering soils (see preceding chapter). The lead content of resulting soil particulate forms is often inversely proportional to particle size, owing to increasing surface to volume ratio with decreasing particle size. Larger surface areas permit more lead binding for a given mass. A second factor is the variability with particle size of lead exposures for children contacting leaded soils (Mushak, 1991). The smaller the lead-containing soil particle, the higher the propensity for adhering to hands and the higher the likelihood of sticking to clothing, shoes, etc. for transport indoors and later potential ingestion (see later chapters).

Bulk soil samples which have not been sieved into different size fractions have the potential to underestimate lead content relative to those smaller particle fractions which have the higher lead exposure potential. That is, larger particles will have lower lead content but may contribute substantially to sample mass. Other mass determination concerns include the need to exclude large-sized detritus and organic matter, since the latter contributes to sample mass but has questionable relevance as a lead exposure medium.

There are a variety of field and laboratory analytical methods for soil lead measurement, depending on the type of analysis and its purposes in a given evaluation. Bulk soil lead measurement refers to measurement of the total lead content of the soil sample. Chemical speciation and micromineralogical studies in the context of human lead exposure variability refer to amounts of specific chemical forms of lead and their geochemical states. These studies are sometimes done in tandem with relative bioavailability testings, i.e., amounts of lead being absorbed under *in vivo* or *in vitro* simulation of *in vivo* conditions (Casteel et al., 2006) with respect to Pb source attribution. Stable isotopic analysis studies deal with the quantitative stratification of lead's stable isotopic composition into the four main stable isotopes: lead-204, lead-206, lead-207, and lead-208 (Gulson et al., 1995, 1997).

Field measurement of bulk soil lead by XRF instruments will typically require confirmation analysis through some randomly selected subset of further testing by some reference technique in the laboratory: AAS, inductively coupled plasma-atomic emission spectroscopy (ICP-AES), or ICP-mass spectrometry (ICP-MS). Other methods are electrochemical in nature, such as ASV and differential pulse polarography. Many soil samples are processed and analyzed directly in the laboratory.

Detection limits for lead in soil matrices have markedly improved over the last several decades owing to improvements in the signal detection systems, e.g., charge-coupled devices in place of photomultiplier tubes.

U.S. EPA (2001) holds ICP-AES or ICP-MS to be the soil lead measurement methods of choice. Detection limits are on the order of 40 parts per billion (ppb) which are quite adequate for an environmental medium where even background, i.e., uncontaminated soil lead, concentrations are on the order of 10–20 ppm.

A number of soil-derived SRMs are available for QA/QC use in the quantitative analysis of lead in soils. The SRM numbers, their corresponding matrix type and lead level (mg/kg, ppm) are 2709, soil, 18.9; 2710, soil, 5532; 2711, soil, 1162; 2586, soil (paint), 432; 2587, and soil (paint) 3242.

Analytical speciation methods, referred to earlier, generally differ from the methods adequate for bulk quantification of lead. As with bulk methods, nonetheless, contaminating levels should not be permitted in interfering amounts. Several lead speciation approaches are X-ray absorption spectrometry, X-ray diffraction, and electron-microprobe microanalyses (U.S. EPA, 2006; Welter et al., 1999).

6.2.4 Analysis of Lead in Dusts

Dust lead arises through transport mechanisms for lead in originating sources, typically lead in paint (Lanphear et al., 1996) and/or ambient air lead deposited onto various surfaces (Adgate et al., 1998). Ambient air lead from both mobile and stationary sources produces interior dust lead (U.S. EPA, 1986a, Ch. 7). Interior dust lead from these input media is of particular concern for childhood lead exposures. Exterior dust lead may also elevate lead exposures from outside toys, play sets, etc. Dust lead can arise from soil lead, which is an environmental medium pathway that receives lead via wet and dry deposition from atmospheric lead or exterior lead paints weathering onto adjacent soils (von Lindern et al., 2003a). A fourth generator of interior dust lead is clothing, shoes, etc. of family members or others who work in leaded settings including battery production and lead secondary smelting and wear their work clothes home. This is termed secondary or “take-home” lead exposure.

Dust sampling for lead uses two different metrics: lead concentration or lead loadings. Each method has its advantages and drawbacks. Determining lead concentrations in dusts typically requires a gravimetric approach, where the mass of lead in collected dusts is quantified from the mass of total dust amounts. Dust lead loadings are done by collecting leaded dusts within a predetermined surface area, e.g., per square meter or per square foot. The lead concentration is influenced by the presence of nonlead materials and house-keeping efficiency differences from site to site. Lead loading per unit area is not affected in this manner (see, e.g., Milar and Mushak, 1982; U.S. EPA, 2001). In addition, leaded dust regulatory standards are currently expressed

soil matrices have markedly improved over to improvements in the signal detection systems in place of photomultiplier tubes.

AES or ICP-MS to be the soil lead measurement limits are on the order of 40 parts per billion for an environmental medium where contaminated soil lead, concentrations are on the

MS are available for QA/QC use in the quantification of lead. The SRM numbers, their corresponding concentrations (g/kg, ppm) are 2709, soil, 18.9; 2710, soil, 432; 2587, and soil (paint) 3242.

Methods, referred to earlier, generally differ from bulk methods, and should not be permitted in interfering with approaches are X-ray absorption spectrometry, electron-microprobe microanalyses (U.S. EPA,

Dusts

Transport mechanisms for lead in originating sources, (Farfel et al., 1996) and/or ambient air lead deposition (Mushak et al., 1998). Ambient air lead from both sources produces interior dust lead (U.S. EPA, 1986a). These input media is of particular concern for interior dust lead may also elevate lead exposures (Mushak et al., 1998). Dust lead can arise from soil lead, which is a pathway that receives lead via wet and dry deposition. Exterior lead paints weathering onto adjacent surfaces. A fourth generator of interior dust lead is workers or others who work in leaded settings and lead secondary smelting and wear their clothing as a secondary or "take-home" lead exposure.

Two different metrics: lead concentration or lead loading. Its advantages and drawbacks. Determining dust lead loading requires a gravimetric approach, where dusts is quantified from the mass of total dust collected. Done by collecting leaded dusts within a pre-defined area, per square meter or per square foot. The lead loading is the presence of nonlead materials and housekeeping practices from site to site. Lead loading per unit area is expressed, e.g., Milar and Mushak, 1982; U.S. EPA, 1995. Regulatory standards are currently expressed

as loadings per square foot. However, expressing lead content as a loading per unit area makes it difficult to draw comparisons with lead in diverse other media where concentration is expressed, and it is relatively difficult to use lead dust loading for lead exposure modeling of human exposure populations (Leggett, 1993; Mushak, 1998; O'Flaherty, 1998).

Surface or media sampling for dust lead is as complex as it is for lead in paint, owing to the number of sampling variables. Dust by its nature is relatively variable mass-wise as a lead-bearing environmental medium compared to lead in paint or lead in soil. For example, the amount of dust accumulation or lead accumulation in dusts is significantly affected by the residence times of the dusts. Housekeeping practices will affect dust sampling measurements within a given home and between homes for samplings of dusts in active contact areas. Surfaces that are relatively undisturbed over extended periods of time—in attics, basements, inaccessible living area surfaces—provide a longer lead accumulation record than areas routinely cleaned.

Other parameters have to do with the purposes of the dust sampling, especially of residential unit exteriors. Dust wipe sampling procedures as part of lead-abatement guidelines or requirements of the U.S. EPA (2001) and U.S. HUD (1995, 1999) are explicitly prescribed. Regulatory lead dust testing to indicate current levels of lead contamination for purposes of human health risk assessment or for setting cleanup levels at Superfund and other contaminated sites are similarly prescribed (von Lindern et al., 2003b). Research projects dealing with dust lead measurements, by contrast, are not as constrained by regulation as by requirements of the study, for example, determination of dust lead loadings versus dust lead concentrations.

Interior paint lead dusts to be sampled in the context of regulatory requirements for either carrying out lead paint hazard risk assessment or for determining the efficacy of lead paint abatement or interim controls are described in such treatises as the 1995 U.S. HUD guidelines for lead paint. Dust lead sampling inside residences with leaded paint surfaces is best done in areas commonly contacted by young children, especially infants and toddlers. These include play areas within rooms, high-traffic commons areas such as hallways, or, particularly important, surfaces beneath windows and window components with leaded paint. Especially problematic for lead exposures are those windows that are frequently opened and closed, and that young children frequently contact.

As set forth in U.S. HUD (1995), dust sampling by use of surface wipes is preferred on the basis of ease of use, relative cost, and a generally favorable performance record for routine samplings. For example, children's blood lead levels and dust wipe lead content in the same units are well correlated (Farfel et al., 1994; Lanphear et al., 1995). Certain vacuum collection approaches can also be used with trained operators in research studies. Measuring dust lead loading from hard surfaces is more reliable than from complex surfaces such as upholstery or carpeting (Ewers et al., 1994).

One can collect dust samples as grab samples or through compositing. In the latter case, at least three subsamples for a composite are recommended where surfaces are relatively similar. Single, grab wipe sampling should be done where a particular surface is not similar to others or where surface deterioration is quite pronounced. That is, it is a surface producing a "hot spot" dust lead location. Compositing, furthermore, should be component- and area-specific. Bare floor samplings should not be combined with carpeted surface collections. Baseboard wipes should not be combined with window sill/trough wipes, etc. Multiple surface wipings with the same wipe should never be done. Each surface should use a new wipe each time. Recommended testing areas in residences occupied by children would include principal playroom, kitchen, bedroom of the youngest child, and bedroom of the second youngest. Dust sampling for multiunit residences expands on the above protocol for single units mainly in adding several common area samples.

Dust sampling for evaluation of residences as part of hazardous waste site evaluations as occur in Superfund activities entails determination of dust lead concentration, usually by vacuum collection through use of vacuum cleaners (von Lindern et al., 2003b) or, preferably, filter collection units attached to vacuum sources.

Laboratory analysis of dust samples employs methods similar to those for measuring lead in soils. However, the total amounts of dust mass sampled for analysis are typically much lower than for soil lead, where soil typically is available in large amounts and sample mass is not problematic. Dust lead levels, on the other hand, tend to track higher at a given testing site than associated soil lead levels. On balance, a more sensitive measurement method is desirable. Flameless AAS and ICP-MS provide both the sensitivity and specificity for acceptable dust lead bulk measurements. Speciation of lead in dusts generally employs methodology similar to that for lead speciation in soils (see earlier).

There are three SRMs that are available from NIST for QA/QC assessment in the laboratory for dust lead measurements (SRM #, matrix, and lead level, ppm): 1649a, urban dust, 12,400; 2583, indoor dust, 85.9; 2584, indoor dust, 9,761.

6.2.5 Analysis of Lead in Diets

Sampling and analysis of lead in diets of human populations require taking account of some characteristics of lead in human diets. First, any given dietary item will have relatively low amounts of lead but the amounts consumed daily can lead to relatively high total lead intakes. Second, while human populations all receive some fraction of their lead exposures from their diets, the size of the fractional intakes and uptakes of lead from diet will vary with such factors as the subset of the population, the amount of the centralized

ab samples or through compositing. In
 les for a composite are recommended.
 Single, grab wipe sampling should be
 similar to others or where surface dete-
 , it is a surface producing a "hot spot"
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 should not be combined with carpeted
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 dust sampling for multiunit residences
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of residences as part of hazardous waste
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Diets

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 es and uptakes of lead from diet will vary with
 the population, the amount of the centralized

versus local food supplies, dietary lead content over time, etc. Adults have a
 different dietary pattern than school-age children, and school-age children
 have a different dietary mix than early infants.

Third, populations in industrialized countries, particularly the United
 States, receive food through a largely centralized food distribution system
 which simplifies somewhat food staple sampling protocols on a population
 or national basis. However, there are exceptions such as those who grow and
 consume their own garden crops or purchase from local farmers' markets.
 Rural residents are more apt to have a higher fraction of local foods than
 residents of large cities.

Lead in diets presents potential exposures for humans in a myriad of
 ways and through complex pathways. Individuals in the United States largely
 rely on a centralized food distribution system but the huge diversity of com-
 ponents of that food system and diversity of food consumption patterns
 across communities and individuals make quantification of dietary lead
 exposure a special challenge. An added factor is time. The relative contribu-
 tion of dietary lead to total lead intakes for human populations, especially in
 the United States, has markedly changed over time. This means that tabula-
 tions of dietary lead levels given later in this chapter have to take account of
 this overall change. Lead intakes also differ over time as a result of changes
 in dietary patterns for reasons including concerns over relative health value
 of particular dietary components, e.g., avoiding obesity and associated
 morbidity.

Sampling methods for determining dietary lead levels range from specific
 approaches—determining dietary lead intakes of individuals at the one
 extreme—to national or international evaluations of the central food supply.
 Galal-Gorchev (1991) described the international dietary lead surveys done
 in the 1980–1988 period under the auspices of the United Nations
 Environmental Program's (UNEP's) Global Environmental Monitoring
 System (GEMS-Food, 1991). Galal-Gorchev reported lead level results for
 25 countries in the GEMS-Food effort, including those for the United States.
 Such samplings are mainly focused on adults in the population, with less
 attention paid to children's dietary lead intakes across countries. However,
 Galal-Gorchev does tabulate the GEMS-Food network's results for lead
 intakes of children up to 12 years of age for 13 countries.

The U.S. dietary lead picture has been evaluated by the U.S. Food and
 Drug Administration (FDA) with varying intensity since the early 1960s,
 largely in the form of the Total Diet Study (TDS). The TDS evaluates nutri-
 ents, pesticides, and other environmental contaminants in the U.S. food sup-
 ply. For a history of the TDS, see Adams (1991). Since the early 1980s, lead
 in diet has been reported annually through the market-basket approach under
 the aegis of the TDS program (Bolger et al., 1991; Pennington, 1983;
 Pennington and Gunderson, 1987). Currently, the TDS approach categorizes
 representative U.S. diets through eight age-sex groups gathered from four

geographic areas of the country and for 12 commodity groups. The 234 subgroups within the 12 commodity groups in turn represent up to 5,000 different foods (Adams, 1991). The TDS approach also relies on data from the U.S. Department of Agriculture's (USDA's) National Food Consumption Survey (NFCS) for both selection of food types and translation of data for lead in diet components into daily dietary lead intakes (Adams, 1991).

Determining lead intakes and exposures for individuals as part of some research purpose will require not general but specific lead intakes. Pao (1989) described four sampling methods for individual dietary assessments, whatever the substances being measured. Two were retrospective in nature and include the recall of past food intake over the last 24 hours or some other time interval and the recall of past usual intake as might be recapitulated in a dietary history. The remaining two were prospective, involving an ongoing record of dietary intake of food components or using duplicate diet food intakes. Of these, the 24-hour food recall is probably the approach which serves for current intakes of dietary lead in either large groups being studied or those for whom the other approaches would not be feasible.

Lead in diet occurs at lower concentrations than in a number of other media, e.g., soils and dusts. Measurement methods therefore require high sensitivity for detection. Lead levels were originally measured by conventional AAS, but now there is more use of bulk sample ashing and Flameless AAS. Capar (1991) described a food lead quantitation limit of 20 ppb.

6.2.6 Analysis of Lead in Drinking Water

Transport of deposited ambient air and soil lead to surface water and groundwater was described in the previous chapter. This chapter is principally concerned with these waters to the extent they serve as drinking water sources for human populations. Human populations typically get their drinking water from individual wells in rural areas or through public water supply distribution systems in suburban and urban locales. Neither well water aquifers nor surface water supplies, e.g., reservoirs, are significant sources of those amounts of lead that enter residential or public tap water. Rather, the distribution system and/or residential/public site tap water plumbing systems are the main sources adding lead to the water. Of these two components of the tap water chain of distribution, household plumbing with leaded connections or fittings is more often the culprit.

Tap water lead is regulated by the U.S. EPA through an action level for the part of the system water in household plumbing and a regulatory maximum contaminant level (MCL) that applies to water leaving water treatment plants. The former is a level of 15 ppb for no more than 10% of cumulatively sampled water testings in a community and the enforceable MCL is 5 ppb. The 1991 EPA lead and copper drinking water rule spells out details for tap water sampling (42 USC §300f; 40 CFR Part 141).

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Concentrations than in a number of other measurement methods therefore require high levels were originally measured by conventional use of bulk sample ashing and Flameless food lead quantitation limit of 20 ppb.

Drinking Water

Air and soil lead to surface water and groundwater. This chapter is principally concerned with how they serve as drinking water sources. Populations typically get their drinking water from rivers or through public water supply distribution systems in urban locales. Neither well water aquifers nor reservoirs, are significant sources of those in private or public tap water. Rather, the distribution of lead in public tap water plumbing systems are the water. Of these two components of the household plumbing with leaded connections are of concern.

The U.S. EPA through an action level for household plumbing and a regulatory maximum that applies to water leaving water treatment plants. The action level is 5 ppb for no more than 10% of cumulatively sampled units and the enforceable MCL is 5 ppb. The drinking water rule spells out details for tap water (40 CFR Part 141).

In general, two types of water samples may be gathered, standing tap water and flushed samples. The former allows evaluation of the lead level in tap water that comes from both household plumbing leaching and lead in water from the distribution system. The latter provides both the distribution contribution to tap water lead and, by difference from the stagnant sampling protocol, individual household-specific plumbing contributions.

Lead in tap water can occur in either fully soluble form or a mixture of soluble and particle-bound, suspended lead. If filtered samples are used, the lead in the insoluble fraction is not accounted for although it can contribute to lead exposures. The latter is of relatively more significance for individual or community wells than for water being distributed from public treatment plants in relatively high-population communities. However, fine particles of lead soldering in household plumbing in suburban and urban areas can contribute to water lead intakes and need attention. In regulatory assessments, such as Superfund risk assessment, total tap water lead, i.e., use of unfiltered samples, is recommended (U.S. EPA, 1989).

6.3 CONCENTRATIONS OF LEAD IN ENVIRONMENTAL MEDIA PRODUCING EXPOSURES

Lead-contaminated environmental media differ in their relative significance for human lead exposures. The first difference is who among human populations are exposed to the Pb in the matrix. Dust and soil lead concentrations are highly significant for lead exposures of young children, especially infants and toddlers as discussed in the following chapter. Nonoccupational adult exposures mainly involve lead in air and diet. In some cases, both children and adults may sustain drinking water lead exposures. Workers are exposed to workplace lead in facilities such as lead smelters and lead-acid battery operations through both inhalation and ingestion, the latter through contact with settled lead dusts. Occupational exposures are not addressed *per se* in this monograph and are mainly noted in the context of secondary or "take-home" lead exposure of workers' families, especially their young children. This type of lead contact occurs through workplace dusts brought home on clothing, shoes, work items, etc.

A second significant factor concerns both the relative amounts of environmental media contamination from lead and the relative amounts of the medium contacted by exposed populations. Both lead intake scenarios can be important, as seen in the next chapter on human exposures. A third broad characteristic has to do with the physical, chemical, physicochemical, and geochemical nature of the element present in some medium. These characteristics affect the extent to which lead levels in some medium are absorbed into the bloodstream.

Lead-containing environmental media and lead concentrations in the media are mainly presented and tabulated in this chapter for ambient air

lead, soil lead, interior and exterior dust lead, dietary lead, and lead in tap water. Lead levels in other, idiosyncratic sources of lead are provided only in brief summary.

Concentrations of lead in the various environmental media described in this section are presented for extended periods. The available data that meet minimal statistical and measurement criteria generally only extend from the late 1960s/early 1970s to the present. The purposes of a wide temporal look at environmental lead concentrations are several. First, the nature of lead as an accumulating contaminant in the bodies of human populations requires an appreciation of the amounts of environmental lead that existed in past decades. As noted earlier, lead levels in media have been changing, mainly downward, so that current human body lead burdens are only partially quantifiable from current lead intakes into body compartments. Secondly, the use of predictive, biokinetic models of human lead exposures for simulating lifetime lead exposures requires knowledge of lead intakes from the earliest periods of life.

6.3.1 Concentrations of Lead in Ambient Air

Prehistoric/Natural Levels in Ambient Air

Prehistoric or "natural" global levels of lead in ambient air are not currently measureable. Only projected estimates of early concentrations can be done. One approach has been to assume that prehistoric levels approximate lead levels measured in the most remote areas of the world using scrupulously rigorous methodologies for both sampling and measurement (see Table 6.7). Maenhaut et al. (1979) measured a value of $7.6 \times 10^{-5} \mu\text{g}/\text{m}^3$ at the South Pole, while Settle and Patterson (1982) recorded an Eniwetok atoll level of $1.7 \times 10^{-4} \mu\text{g}/\text{m}^3$ and a similar value of $1.5 \times 10^{-4} \mu\text{g}/\text{m}^3$ was recorded by Davidson et al. (1981) at a Greenland site. A second approach, that of Nriagu (1979) and Settle and Patterson (1980), entailed calculations of air Pb levels derived from estimating natural global emissions and dispersion of the emissions into selected tropospheric volumes, and assuming a residence time of 10 days. The two corresponding estimates are $2.6 \times 10^{-4} \mu\text{g Pb}/\text{m}^3$ (Nriagu, 1979) and $2.1 \times 10^{-5} \mu\text{g Pb}/\text{m}^3$.

The U.S. EPA, in its 1986 Air Quality Criteria for Lead document, selected the figure of $5 \times 10^{-5} \mu\text{g Pb}/\text{m}^3$ as the most reasonable depiction of the various estimating outcomes for purposes of subsequently calculating a background air lead value.

Ambient Air Pb Levels in the Modern Era

Ambient air Pb levels in the United States or elsewhere were not recorded or reported in any systematic way until the late 1950s. The establishment of the National Air Surveillance Network (NASN) in the United States was typical

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Lead in Ambient Air

in Ambient Air

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of such efforts. By the late 1970s, the NASN sites numbered 300 urban and 30 nonurban monitoring units. Since then, the numbers have declined with the phasing out of lead additives in gasoline (see later). However, other U.S. surveillance networks have been established and remain operational mainly through the U.S. EPA. These current networks measure different characteristics of ambient air Pb.

The total suspended particulate (TSP) measuring networks, managed by local and state agencies, currently numbering about 250 sites, measure lead via official Federal methods (40 CFR: Part 40, Appendix G) in particulate matter sized up to $30 \mu\text{m}$. The retention of the TSP networks is driven by the regulatory structure of the previous and current ambient air lead primary and secondary standards. Locations of these sites are depicted in U.S. EPA (2006, Ch. 3).

Beginning in 2000, the Speciation Trends Network (STN) operated to quantify the lead content of particulate matter $2.5 \mu\text{m}$ or less ($\text{PM}_{2.5}$). Active $\text{PM}_{2.5}$ sites, as of this writing, number about 50 and look at long-term trends in the United States. These are augmented with 150 additional sites (U.S. EPA, 2006, Ch. 3). These STN sites are principally oriented toward urban areas.

The remaining two U.S. networks are the Interagency Monitoring of Protected Invisible Environments (IMPROVE) system and the National Air Toxics Trends Stations (NATTS). The IMPROVE network, as the label implies, deals with lead and other contaminants in rural areas measured in the $\text{PM}_{2.5}$ fraction. The National Park Service manages the principal 110 rural IMPROVE sites with shared management through other parties for 80 more sites that are a mix of urban and rural. The NATTS network is the newest of the group of networks, coming on stream in 2004, and is a set of 23 sites for urban and some rural areas. Lead measurement is confined to particle sizes of $10 \mu\text{m}$ or less.

The earliest U.S. data gathering, before 1966, had problematic results according to the U.S. EPA (1977). Because of that finding, data recorded in this chapter begin with reports for 1966. Concerns about earlier data included factors such as analytical inadequacy, statistical design problems, and sampling issues. Similar concerns about reliability of earlier air lead data elsewhere in the world limited international monitoring in general. As described earlier, adequate and sensitive air particulate sampling and laboratory measurement methods were still in development.

Three periods are covered in tabulating U.S. ambient air lead levels: 1966–1974, 1975–1984, and 1985 to the present. The first interval captures the increase and peak in air lead from mobile (leaded gasoline use) and stationary (e.g., industrial, fossil fuel, and waste combustion) facilities. The second period overlaps the regulatory phasing down period for lead emissions to the air, while the final period of 1985 to the present represents the current picture for air lead levels. As noted earlier, inclusion of earlier periods with their much higher atmospheric lead burdens is intended to help quantify

TABLE 6.1 Percentage of NASN Stations Reporting Urban Air Lead Data at Indicated Air Lead ($\mu\text{g Pb/m}^3$) Intervals^{a,b}

Year	Air Pb Interval ($\mu\text{g/m}^3$)				
	<0.5	0.5–0.99	1.0–1.9	2.0–3.9	4.0–5.3
1966	9	42	42	6	—
1967	3	32	55	7	—
1968	9	45	36	6	1
1969	2	25	57	12	1
1970	5	33	50	9	1
1971	—	21	58	19	1
1972	9	37	47	7	0
1973	15	55	26	3	1
1974	15	53	29	3	0
1966–1974 (Mean)	8	38	45	8	1

^aAdapted from U.S. EPA (1977, Ch. 7).^bPercentages of reporting stations within the air Pb intervals indicated sum to approximately 100%.

estimates for atmospheric lead inputs to long-lived reservoirs of anthropogenic lead—soils, dusts, sediments—and to assist in modeling lifetime lead exposures in older populations exposed to those earlier levels. The topic of cumulative lead exposures of human populations in developed, industrialized societies is developed later in this text (Mushak and Mushak, 2000).

Table 6.1 provides a useful distribution tally of urban NASN stations reporting U.S. urban air lead ranges ($\mu\text{g/m}^3$) within the indicated intervals and over the period 1966–1974. The figures are adapted from U.S. EPA (1977, Ch. 7).

As expected in this period of peak leaded gasoline use and heavy emissions from stationary sources such as smelters, the highest air lead readings occur for the intervals representing traffic densities and/or stationary site emissions. The two highest air lead ranges largely capture areas of point source lead emissions, with the highest air lead range covering one emission site.

Table 6.1 shows that the highest percentage of stations reporting within the air lead range 0.50–0.99 $\mu\text{g/m}^3$, 55%, occurred in 1973. The highest percentage, 57%, of all NASN monitoring sites for the next highest air lead interval of 1.0–1.9 $\mu\text{g/m}^3$ was reported in 1969. For the next highest air lead

TABLE 6.1 Percentage of Nonurban NASN Stations Reporting Air Lead Data at Indicated Air Lead ($\mu\text{g Pb}/\text{m}^3$) Intervals^{a,b}

Air Pb Interval ($\mu\text{g}/\text{m}^3$)			
0.5–0.99	1.0–1.9	2.0–3.9	4.0–5.3
42	42	6	—
32	55	7	—
45	36	6	1
25	57	12	1
33	50	9	1
21	58	19	1
37	47	7	0
55	26	3	1
53	29	3	0
38	45	8	1

^aNumber of reporting NASN stations varies across the air Pb intervals indicated sum to approximately

inputs to long-lived reservoirs of anthropogenic lead—and to assist in modeling lifetime lead exposure to those earlier levels. The topic of human populations in developed, industrialized areas is discussed in this text (Mushak and Mushak, 2000).

The distribution tally of urban NASN stations reporting air lead ($\mu\text{g}/\text{m}^3$) within the indicated intervals is presented in Table 6.1. The figures are adapted from U.S. EPA

of peak leaded gasoline use and heavy emissions from sources such as smelters, the highest air lead readings were reported from monitoring sites with the highest air lead range covering one

highest percentage of stations reporting within the range 0.5–0.99 $\mu\text{g}/\text{m}^3$, 55%, occurred in 1973. The highest percentage of reporting sites for the next highest air lead range was reported in 1969. For the next highest air lead

TABLE 6.2 Percentage of Nonurban NASN Stations Reporting Air Lead Data at Indicated Air Lead ($\mu\text{g Pb}/\text{m}^3$) Intervals^{a,b}

Year	Air Lead Interval ($\mu\text{g}/\text{m}^3$)			
	<0.03	0.03–0.099	0.10–0.19	0.20–0.45
1966	—	52	32	16
1967	—	35	50	10
1968	5	75	20	—
1969	—	52	43	5
1970–1971	—	—	70	30
1972	29	12	26	33
1973	39	31	26	4
1974	19	31	38	12
1966–1974	15	36	35	14

^aAdapted from U.S. EPA (1977, Ch. 7).

^bNumber of reporting NASN stations varies across the air Pb intervals.

range, 2.0–3.9 $\mu\text{g}/\text{m}^3$, the highest percentage, 19%, occurred in 1971. For the time period 1966–1974, the average percentage distribution of reporting sites for the ranges <0.5, 0.5–0.99, 1.0–1.9, 2.0–3.9, and 4.0–5.3 $\mu\text{g}/\text{m}^3$ were 8%, 38%, 45%, 8%, and 1% respectively. The highest percentage, 45%, was reported in the air lead range of 1.0–1.9 $\mu\text{g}/\text{m}^3$, followed closely by 38% for the range 0.5–0.99 $\mu\text{g}/\text{m}^3$. Combined, 83% of all stations reported air Pb in the range 0.5–1.9 $\mu\text{g}/\text{m}^3$.

Analogous percentage distribution calculations for reporting NASN stations in nonurban areas are presented in Table 6.2. Air Pb levels in the two higher ranges of 0.03–0.099 and 0.10–0.19 $\mu\text{g}/\text{m}^3$ account for the majority of the reporting site levels up to about 1971, followed by a decline in later years. The highest percentage of reporting stations were generally for air Pb values 0.030–0.099 $\mu\text{g}/\text{m}^3$ followed by the range of 0.10–0.19 $\mu\text{g}/\text{m}^3$. For the years 1966–1974, the average percentages of sites reporting various air Pb ranges showed 36% and 35% for the intervals 0.030–0.099 and 0.10–0.19 $\mu\text{g}/\text{m}^3$.

Table 6.3 presents additional data on the descriptive statistics for U.S. urban air lead measured quarterly in the 1970–1974 time frame in the form of means, maxima, and percentile distributions of air lead concentrations. Both arithmetic (1.19, 1.23, 1.13 $\mu\text{g}/\text{m}^3$) and geometric (0.99, 1.00, 0.03 $\mu\text{g}/\text{m}^3$) means were similarly elevated from 1970 to 1972, respectively, followed

TABLE 6.3 Mean, Percentile Distribution, and Maximum Urban Air Lead Quarterly Measurements from Urban Stations, 1970–1974^{a,b}

Year	Arithmetic Mean ($\mu\text{g}/\text{m}^3$)	Geometric Mean ($\mu\text{g}/\text{m}^3$)	Maximum ($\mu\text{g}/\text{m}^3$)	Percentile Distribution				
				10	50	70	95	99
1970	1.19	0.99	5.83	0.47	1.05	1.37	2.59	4.14
1971	1.23	1.00	6.31	0.42	1.01	1.42	2.86	4.38
1972	1.13	0.93	6.88	0.46	0.97	1.25	2.57	3.69
1973	0.92	0.76	5.83	0.35	0.77	1.05	2.08	3.03
1974	0.89	0.75	4.09	0.36	0.75	1.00	1.97	3.16

^aAdapted from U.S. EPA (1977, Ch. 7).^bNumber of quarterly composites varies.**TABLE 6.4** Mean, Maximum, and Percentile Distribution of Nonurban Air Lead Quarterly Measurements from Nonurban Stations, 1970–1974^{a,b}

Year	Arithmetic Mean ($\mu\text{g}/\text{m}^3$)	Geometric Mean ($\mu\text{g}/\text{m}^3$)	Maximum ($\mu\text{g}/\text{m}^3$)	Percentile Distribution				
				10	50	70	95	99
1970	0.09	0.04	1.47	0.00	0.00	0.00	0.38	0.63
1971	0.05	0.01	1.13	0.00	0.00	0.00	0.20	0.78
1972	0.14	0.09	1.05	0.01	0.11	0.17	0.39	0.95
1973	0.11	0.07	0.94	0.02	0.06	0.13	0.39	0.70
1974	0.11	0.08	0.53	0.01	0.09	0.14	0.32	0.50

^aAdapted from U.S. EPA (1977).^bNumber of quarterly composites varies.

by onset of declines in 1973 and 1974. Median (50th percentile) air lead levels showed a similar comparative relationship, at 1.05, 1.01, and 0.97 $\mu\text{g}/\text{m}^3$ for 1970–1972, respectively, followed by declines to 0.77 and 0.75 $\mu\text{g}/\text{m}^3$ for the later 2 years. Maxima for these years were 5.83, 6.31, 6.88, 5.83, and 4.09 $\mu\text{g}/\text{m}^3$.

Descriptive statistics are similarly provided for nonurban air lead values in the 1970–1974 time period, in Table 6.4. Here, the picture is less clear across the 5 years of measurement, giving no definitive trend. Results also reflect the lower number of monitoring stations and variability in the number

Distribution, and Maximum Urban Air Lead
Urban Stations, 1970–1974^{a,b}

Maximum ($\mu\text{g}/\text{m}^3$)	Percentile Distribution				
	10	50	70	95	99
5.83	0.47	1.05	1.37	2.59	4.14
6.31	0.42	1.01	1.42	2.86	4.38
6.88	0.46	0.97	1.25	2.57	3.69
5.83	0.35	0.77	1.05	2.08	3.03
4.09	0.36	0.75	1.00	1.97	3.16

and Percentile Distribution of Nonurban Air
from Nonurban Stations, 1970–1974^{a,b}

Maximum ($\mu\text{g}/\text{m}^3$)	Percentile Distribution				
	10	50	70	95	99
1.47	0.00	0.00	0.00	0.38	0.63
1.13	0.00	0.00	0.00	0.20	0.78
1.05	0.01	0.11	0.17	0.39	0.95
0.94	0.02	0.06	0.13	0.39	0.70
0.53	0.01	0.09	0.14	0.32	0.50

and 1974. Median (50th percentile) air lead
comparative relationship, at 1.05, 1.01, and
respectively, followed by declines to 0.77 and
s. Maxima for these years were 5.83, 6.31,

ilarly provided for nonurban air lead values
in Table 6.4. Here, the picture is less clear
ent, giving no definitive trend. Results also
monitoring stations and variability in the number

TABLE 6.5 Average Air Pb and Particle Size Distributions for Six Major
U.S. Cities in 1970^a

City	Average Annual Pb Level ($\mu\text{g}/\text{m}^3$)	Average Particle Size MMD (μm) ^b	% Particles $\leq 1 \mu\text{m}$
Chicago, IL	3.2	0.68	59
Cincinnati, OH	1.8	0.48	72
Denver, CO	1.8	0.50	70
Philadelphia, PA	1.6	0.47	62
St. Louis, MO	1.8	0.69	62
Washington, DC	1.3	0.42	74

^aAdapted from U.S. EPA (1977, Ch. 7).

^bMMD = mass median diameter.

of quarterly composites. During the period 1970–1974, the arithmetic mean values were 0.09, 0.05, 0.14, 0.11, and 0.11 $\mu\text{g}/\text{m}^3$, respectively. Corresponding geometric mean values were 0.04, 0.01, 0.09, 0.07, and 0.08 $\mu\text{g}/\text{m}^3$, respectively, while maxima for the 5 years were 1.47, 1.13, 1.05, 0.94, and 0.53 $\mu\text{g}/\text{m}^3$, respectively. Unlike the measures of central tendency, the maxima show a downward trend from 1970 to 1974.

Mean values of air lead concentration were on the order of 10- to 20-fold lower for nonurban versus urban monitoring reports, but maxima for the two categories varied less, about fourfold lower. The latter likely reflects the principal contributor to nonurban air lead, point source contributions such as lead smelters. The nonurban sites are typically located in more remote areas, in the Midwest and West.

Table 6.5 presents mean air lead values in 1970 for six major U.S. cities as well as data for lead-bearing particle sizes. The mean air lead concentrations for Chicago, Cincinnati, Denver, Philadelphia, St. Louis, and Washington, DC, were 3.2, 1.8, 1.8, 1.6, 1.8, and 1.3 $\mu\text{g}/\text{m}^3$, respectively. Average sizes of measured air particulate ranged from 0.42 to 0.69 μm average MMD, while the percentages of particles $\leq 1 \mu\text{m}$ ranged from 59% to 74%. The fraction of lead in the small particle fraction indicates both a size with longer geographic reach and a size that is respirable by human populations.

Table 6.6 depicts ambient air lead statistics gathered by the U.S. EPA for 1973; the Seven-City Study of U.S. air lead for commercial, industrial, and residential monitoring sites. The U.S. cities were Chicago, Cincinnati, Houston, Los Angeles, New York, Philadelphia, and Washington, DC. Data reported are broken into monthly mean, minimum, and maximum values.

TABLE 6.6 U.S. Ambient Air Lead Statistics^a for the Seven-City Study: Chicago, Cincinnati, Houston, Los Angeles, New York, Philadelphia, and Washington, DC^b

City	Site Type ^c	Monthly Level ($\mu\text{g}/\text{m}^3$) ^d		
		Mean	Minimum	Maximum
Chicago, IL	C	4.5	2.5	6.7
	I	3.7	1.7	7.0
	R	3.3	2.1	4.7
Cincinnati, OH	C	2.0	1.3	3.1
	I	2.2	1.2	2.8
	R	1.5	0.8	2.6
Houston, TX	C	1.9	1.1	3.1
	I	—	—	—
	R	1.0	0.6	1.8
Los Angeles, CA	C	0.2	0.1	0.3
	I	—	—	—
	R	0.2	0.1	0.3
New York, NY	C	—	—	—
	I	—	—	—
	R	1.4	1.0	1.8
Philadelphia, PA	C	2.8	1.9	3.9
	I	2.2	1.5	3.0
	R	1.3	0.8	1.9
Washington, DC	C	1.4	3.1	2.0
	I	—	—	—
	R	1.5	1.1	1.8

^aFrom Tepper and Levin 1975. As discussed and cited in U.S. EPA (1977, Appendix C).

^bAdapted from U.S. EPA (1977, Appendix C).

^cC = commercial; I = industrial; R = residential.

^dMean of multiple values for site types, mainly 12 months of data.

Some comparisons cannot be made for a site category across cities because data were not available. Nonetheless, all stations for these cities reported residential area monitoring measurements, while all but one city reported commercial district air lead concentrations.

Lead Statistics^a for the Seven-City Study:
Los Angeles, New York, Philadelphia, and

	Monthly Level ($\mu\text{g}/\text{m}^3$) ^d		
	Mean	Minimum	Maximum
	4.5	2.5	6.7
	3.7	1.7	7.0
	3.3	2.1	4.7
	2.0	1.3	3.1
	2.2	1.2	2.8
	1.5	0.8	2.6
	1.9	1.1	3.1
	—	—	—
	1.0	0.6	1.8
	0.2	0.1	0.3
	—	—	—
	0.2	0.1	0.3
	—	—	—
	—	—	—
	1.4	1.0	1.8
	2.8	1.9	3.9
	2.2	1.5	3.0
	1.3	0.8	1.9
	1.4	3.1	2.0
	—	—	—
	1.5	1.1	1.8

Source: Data collected and cited in U.S. EPA (1977, Appendix C).

^a Data are for Los Angeles, New York, Philadelphia, and

^b Confidential.
^c Data are for mainly 12 months of data.

made for a site category across cities because, regardless of the city, all stations for these cities reported residential measurements, while all but one city reported commercial measurements.

The city reporting the highest residential and commercial district values was Chicago. Mean, minimum, and maximum residential air Pb concentrations were 3.3, 2.1, and 4.7 $\mu\text{g}/\text{m}^3$, respectively. Los Angeles provided the lowest air lead results, with the mean, minimum, and maximum concentrations being 0.2, 0.1, and 0.3 $\mu\text{g}/\text{m}^3$, respectively. Mean data for the remaining five cities in descending order were 1.5, 1.5, 1.4, 1.3, and 1.0 $\mu\text{g}/\text{m}^3$ for Washington, DC, Cincinnati, New York, Philadelphia, and Houston, respectively. The air lead values reflect mobile lead emissions from vehicular consumption of leaded gasoline but clearly other factors are operative. The relative urban character and population densities of each city's residential areas arguably play a role but multiple emission sources of airborne lead contribute. For example, residential air lead values for Chicago are several-fold higher than those of New York, despite similar population density or even greater multiple-unit residential density in New York. Chicago data may reflect both vehicular and industrial emissions, a more heterogeneous mix of multiple lead emission sources to the atmosphere.

The U.S. air lead levels are presented here for later periods as well. These later periods generally reflect decline in consumption of lead and consequently production of lead (U.S. EPA, 1986a, 2006). There was the major decline in leaded gasoline consumption due to regulatory restrictions leading to, first, a phasedown of the amounts of antiknock additive permitted in fuels in the 1980s and early 1990s, followed by an eventual phaseout. The numbers of point source lead emissions from primary and secondary lead smelters markedly declined as well.

Such changes are captured in Table 6.7, which depicts urban air lead levels for downtown sites in nine major U.S. cities from 1975 to 1984. Most of these cities showed air lead quarterly averages on the order of 1.0–2.0 $\mu\text{g}/\text{m}^3$ for the period 1978–1979, followed by declines of 50–70% or more from 1980 to 1984. Marked declines in some cities to 15–20% of 1975 figures were reported by 1984.

The tabulated air lead figures for U.S. areas were approximated globally, especially in major cities outside the United States. In a number of cases, cities outside the United States presented figures greater than those in the United States for the period 1975–1984. This difference arose in some significant part from the relatively slower speed of change in lead content of gasoline for non-U.S. vehicular fleets and other lead uses.

Table 6.8 tabulates air lead data for non-U.S. urban areas and rural around the globe. Locations, year(s) of measurement, and reported air lead concentration summaries are given. The earliest of the years monitored for each national locale in Table 6.8 show quite high air lead, compared to U.S. data shown in the earlier tables for about the same time period. Sites in Italy, Saudi Arabia, and Greece had particularly elevated air lead, ranging from 3.2 to 5.5 $\mu\text{g}/\text{m}^3$. In most cases where multiple years of measurement were noted, declines in air lead are seen post-1979. Table 6.8 also shows that size of urban area or

TABLE 6.7 Annualized Air Lead Levels^{a,b,c} ($\mu\text{g}/\text{m}^3$) for Major U.S. Metropolitan Areas, 1975–1984

Year	U.S. Urban Areas Pb ($\mu\text{g}/\text{m}^3$)								
	Boston, MA	New York, NY	Philadelphia, PA	Washington, DC	Detroit, MI	Chicago, IL	Houston, TX	Dallas, TX	Los Angeles, CA
1975	0.93	0.93	—	1.1	0.98	—	2.1	2.8	2.1
1976	—	—	—	1.0	—	—	0.87	0.83	—
1977	0.65	—	1.4	1.4	1.0	—	0.80	1.5	2.1
1978	1.0	1.3	1.3	1.9	—	—	1.1	1.5	1.9
1979	0.70	0.95	1.1	1.7	—	0.60	0.73	0.85	1.0
1980	0.67	—	0.63	—	0.33	0.65	0.45	0.40	0.90
1981	0.35	—	0.43	—	0.30	0.33	0.55	0.45	1.0
1982	1.0	0.53	—	0.23	—	0.33	—	0.55	0.80
1983	0.50	0.35	—	0.23	—	0.38	0.30	0.70	0.70
1984	0.45	0.35	—	0.20	0.15	0.25	0.30	0.38	0.40

^aAdapted from U.S. EPA (1986a, Ch. 7).^bAverage of reported quarterly averages given in U.S. EPA (1986a, Ch. 7).^cFour different types of reporting downtown commercial sites used.

1977	0.65	—	1.4	1.4	1.0	—	0.80	1.5	2.1
1978	1.0	1.3	1.3	1.9	—	—	1.1	1.5	1.9
1979	0.70	0.95	1.1	1.7	—	0.60	0.73	0.85	1.0
1980	0.67	—	0.63	—	0.33	0.65	0.45	0.40	0.90
1981	0.35	—	0.43	—	0.30	0.33	0.55	0.45	1.0
1982	1.0	0.53	—	0.23	—	0.33	—	0.55	0.80
1983	0.50	0.35	—	0.23	—	0.38	0.30	0.70	0.70
1984	0.45	0.35	—	0.20	0.15	0.25	0.30	0.38	0.40

^aAdapted from U.S. EPA (1986a, Ch. 7).
^bAverage of reported quarterly averages given in U.S. EPA (1986a, Ch. 7).
^cFour different types of reporting downtown commercial sites used.

TABLE 6.8 Air Lead Level Reports for Locations Outside the United States: Urban and Rural Measurements for 1975–1984

		Location	
Urban	Year(s) of Measurement	Air Pb Concentration ($\mu\text{g}/\text{m}^3$)	References
Brussels, Belgium	1978	0.50	Roels et al. (1980)
Brussels, Belgium	1979	1.05	Ducoffre et al. (1990)
Other Belgian sites	1983	0.66	
Ottawa, Canada	1975	1.30	NAPS (1971–1976)
Toronto, Canada	1975	1.30	NAPS (1971–1976)
Toronto, Canada	1984	0.45	O'Heany et al. (1988)
Montreal, Canada	1975	2.0	NAPS (1971–1976)
Helsinki, Finland	1983	0.33	Ponka et al. (1991)
Athens, Greece	1979	3.20	Chartsias et al. (1986); Kapaki et al. (1998)
	1982	1.76	
	1984	0.91	
	1974–1979	4.5	
Turin, Italy	1980	3.0	Facchetti (1989)
	1983	5.5	El-Shobokshy (1985)
Stockholm, Sweden	1980	1.20	Elinder et al. (1986)
	1983	0.50	
<i>Sites in Wales</i>			
Queensferry	1984	0.55	Page et al. (1988)
Wrexham		0.21	
Bedwas		0.22	
Port Talbot		0.14	
<i>Cities in France</i>			
Paris	1984–1985	0.44	Del Delumyea and Kalivretenos (1987)
Strasbourg		0.07	
Clermont		0.05	
Orleans		0.11	
Senonches		0.01	

(Continued)

TABLE 6.8 Air Lead Level Reports for Locations Outside the United States: Urban and Rural Measurements for 1975–1984—(cont.)

Rural	Location		References
	Year(s) of Measurement	Air Pb Concentration ($\mu\text{g}/\text{m}^3$)	
Canada	1984	0.10	O'Heany et al. (1988)
Italy	1976–1980	0.33	Facchetti and Geiss (1982)
Belgium	1978	0.37	Roels et al. (1980)

level of industrialization is directly related to air lead content. Paris showed the highest air lead level for French cities, with the lowest value cited for the small town of Senonches. A similar relationship was noted for Wales.

Table 6.9 depicts peak air lead levels (quarterly maximum average, $\mu\text{g}/\text{m}^3$) reported for 1994 and for all major urban areas of the United States with a population of 1 million or more compiled in the 1990 U.S. Census and arranged alphabetically. These 48 urban areas are the metropolitan statistical areas (MSAs), comprising an identified central city and, where indicated, contiguous urban populations. Four did not provide air lead values, leaving a net of 44 sites. Table 6.9, adapted from U.S. EPA (1995), has data for monitoring sites which are directed toward the greatest populations even though EPA records air lead values for nonpopulation monitors as well. This adjustment avoids the use of anomalous values that capture large point source air lead emissions that have minimal impact in terms of numbers of individuals affected.

Of the 44 entries for the largest U.S. MSAs with air lead levels reported in 1994, over half ($N = 25$) reported a peak air lead $\leq 0.05 \mu\text{g}/\text{m}^3$. A total of eight MSAs reported air lead in the range of >0.05 – $0.10 \mu\text{g}/\text{m}^3$, while eight MSAs had peak air lead values of 0.11 – $0.30 \mu\text{g}/\text{m}^3$. Three MSAs exceeded peak air lead of $0.30 \mu\text{g}/\text{m}^3$, with the highest level for all MSAs being $0.89 \mu\text{g}/\text{m}^3$. A majority of the locations (57%) had peak air lead of $0.05 \mu\text{g}/\text{m}^3$ or less. Overall, the 1994 MSAs show peak air lead values that are but a small fraction of typical U.S. values in the 1970s and 1980s.

The U.S. air lead levels largely showed their maximal decline from peak values in the 1960s and early 1970s by 1994, with relatively more modest declines afterwards. However, relative to the picture in 1994, the proportional decline into the 2000–2005 time frame was still measurable.

ports for Locations Outside the United Measurements for 1975–1984—(cont.)

Location	Air Pb Concentration ($\mu\text{g}/\text{m}^3$)	References
	0.10	O'Heany et al. (1988)
	0.33	Facchetti and Geiss (1982)
	0.37	Roels et al. (1980)

ectly related to air lead content. Paris showed
ench cities, with the lowest value cited for the
ilar relationship was noted for Wales.
ad levels (quarterly maximum average, $\mu\text{g}/\text{m}^3$)
major urban areas of the United States with
more compiled in the 1990 U.S. Census and
48 urban areas are the metropolitan statisti-
an identified central city and, where indi-
cations. Four did not provide air lead values,
le 6.9, adapted from U.S. EPA (1995), has
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of 0.11 – $0.30 \mu\text{g}/\text{m}^3$. Three MSAs exceeded
with the highest level for all MSAs being
cations (57%) had peak air lead of $0.05 \mu\text{g}/\text{m}^3$
show peak air lead values that are but a small
the 1970s and 1980s.

ely showed their maximal decline from peak
1970s by 1994, with relatively more modest
relative to the picture in 1994, the propor-
–2005 time frame was still measurable.

TABLE 6.9 Peak Air Lead Levels^a in Major U.S. Urban Areas^{b,c,d}, 1994^e

Location	1990 Population	Air Lead ($\mu\text{g}/\text{m}^3$)
Atlanta, GA	2,833,511	≤ 0.05
Baltimore, MD	2,382,172	0.03
Bergen–Passaic, NJ	1,278,440	0.04
Boston, MA–NH	2,870,669	0.01
Buffalo–Niagara Falls, NY	1,189,288	0.05
Charlotte, NC–Gastonia, NC–Rock Hill, SC	1,162,093	0.03
Chicago, IL ^f	6,069,974	0.10
Cincinnati, OH–KY–IN	1,452,645	0.04
Cleveland–Lorain–Elyria, OH ^g	2,202,069	0.12
Columbus, OH	1,377,419	0.11
Dallas, TX ^h	2,553,362	0.08
Denver, CO	1,622,980	0.07
Detroit, MI	4,382,299	0.07
Fort Worth–Arlington, TX	1,332,053	0.03
Houston, TX	3,301,937	0.01
Indianapolis, IN ⁱ	1,249,822	0.20
Kansas City, MO–KS	1,566,280	0.03
Los Angeles–Long Beach, CA	8,863,164	0.08
Miami, FL	3,192,582	0.01
Middlesex–Somerset–Huntingdon, NJ	1,019,835	0.12
Milwaukee–Waukesha, WI	1,432,149	0.03
Minneapolis–St. Paul, MN–WI	2,464,124	Not available
Nassau–Suffolk, NY	2,609,212	Not available
Newark, NJ	1,824,321	0.30
New Orleans, LA	1,238,816	0.12
New York, NY	8,546,846	0.11
Norfolk–Virginia Beach–Newport News, VA	1,396,107	0.02
Oakland, CA	2,082,914	0.02

(Continued)

TABLE 6.9 Peak Air Lead Levels^a in Major U.S. Urban Areas^{b,c,d}, 1994^e—
(cont.)

Location	1990 Population	Air Lead ($\mu\text{g}/\text{m}^3$)
Orange County, CA	2,410,556	0.04
Orlando, FL	1,072,748	0.00
Philadelphia, PA—NJ ^j	4,856,881	0.49
Phoenix—Mesa, AZ	2,122,101	0.04
Pittsburgh, PA	2,056,705	0.07
Portland—Vancouver, OR—WA ^k	1,239,842	0.27
Providence—Fall River—Warwick, RI—MA	1,141,501	Not available
Riverside—San Bernardino, CA	2,588,793	0.04
Rochester, NY	1,002,410	0.04
Sacramento, CA	1,481,102	0.02
St. Louis, MO—IL ^l	2,444,099	0.06
Salt Lake City—Ogden, UT	1,072,227	0.05
San Antonio, TX	1,302,099	0.03
San Diego, CA	2,498,016	0.02
San Francisco, CA	1,603,678	0.02
San Jose, CA	1,497,577	0.02
San Juan—Bayamon, PR	1,086,376	Not available
Seattle—Bellevue—Everett, WA	1,972,961	0.61
Tampa—St. Petersburg—Clearwater, FL ^m	2,067,959	0.89
Washington, DC—MD—VA—WV	3,000,504	0.04

^aBased on site air lead monitors for MSAs arranged alphabetically.^bQuarterly maximum average, $\mu\text{g}/\text{m}^3$.^cDefined as MSA with population ≥ 1 million.^dPopulation from 1990 census.^eAdapted from U.S. EPA (1995, Table A-12).^fValue for population-directed monitoring.^gLevel for Cleveland itself.^hValue for population-oriented monitoring.ⁱValue for population-oriented monitoring.^jHighest population-oriented monitor result.^kImpact from an industrial source.^lOriginal value impacted by an industrial source; highest reading from population-oriented source used.^mHighest population-oriented monitor result.

TABLE 6.9 Peak Air Lead Levels^a in Major U.S. Urban Areas^{b,c,d}, 1994^e—
(cont.)

Location	1990 Population	Air Lead ($\mu\text{g}/\text{m}^3$)
Orange County, CA	2,410,556	0.04
Orlando, FL	1,072,748	0.00
Philadelphia, PA—NJ ^j	4,856,881	0.49
Phoenix—Mesa, AZ	2,122,101	0.04
Pittsburgh, PA	2,056,705	0.07
Portland—Vancouver, OR—WA ^k	1,239,842	0.27
Providence—Fall River—Warwick, RI—MA	1,141,501	Not available
Riverside—San Bernardino, CA	2,588,793	0.04
Rochester, NY	1,002,410	0.04
Sacramento, CA	1,481,102	0.02
St. Louis, MO—IL ^l	2,444,099	0.06
Salt Lake City—Ogden, UT	1,072,227	0.05
San Antonio, TX	1,302,099	0.03
San Diego, CA	2,498,016	0.02
San Francisco, CA	1,603,678	0.02
San Jose, CA	1,497,577	0.02
San Juan—Bayamon, PR	1,086,376	Not available
Seattle—Bellevue—Everett, WA	1,972,961	0.61
Tampa—St. Petersburg—Clearwater, FL ^m	2,067,959	0.89
Washington, DC—MD—VA—WV	3,000,504	0.04

^aBased on site air lead monitors for MSAs arranged alphabetically.^bQuarterly maximum average, $\mu\text{g}/\text{m}^3$.^cDefined as MSA with population ≥ 1 million.^dPopulation from 1990 census.^eAdapted from U.S. EPA (1995, Table A-12).^fValue for population-directed monitoring.^gLevel for Cleveland itself.^hValue for population-oriented monitoring.ⁱValue for population-oriented monitoring.^jHighest population-oriented monitor result.^kImpact from an industrial source.^lOriginal value impacted by an industrial source; highest reading from population-oriented source used.^mHighest population-oriented monitor result.

als^a in Major U.S. Urban Areas^{b,c,d}, 1994^e —

	1990 Population	Air Lead ($\mu\text{g}/\text{m}^3$)
	2,410,556	0.04
	1,072,748	0.00
	4,856,881	0.49
	2,122,101	0.04
	2,056,705	0.07
	1,239,842	0.27
RI—MA	1,141,501	Not available
	2,588,793	0.04
	1,002,410	0.04
	1,481,102	0.02
	2,444,099	0.06
	1,072,227	0.05
	1,302,099	0.03
	2,498,016	0.02
	1,603,678	0.02
	1,497,577	0.02
	1,086,376	Not available
	1,972,961	0.61
r, FL ^m	2,067,959	0.89
	3,000,504	0.04

^aAs arranged alphabetically.

^bmillion.

^cA-12).

^ding.

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^gresult.

^hial source; highest reading from population-oriented source

ⁱresult.

For example, air lead averages in the Los Angeles area noted by Hui (2002) were 0.015–0.019 $\mu\text{g}/\text{m}^3$ compared to the figure of 0.08 $\mu\text{g}/\text{m}^3$ in 1994, a decline of about 75%.

The most detailed data sets for U.S. national air lead statistics as of this writing are those contained in the Air Toxics Data Archive (2003–2005), a joint effort of the U.S. EPA, state/territorial air pollution officials, and local air control officials (U.S. EPA, 2007a). EPA (2007a) reported air lead in TSP measured at 189 monitoring sites, 140 of which were urban areas. The same material reported the national figures for these 3 years using four different statistical metrics: annual mean, maximum quarterly mean, maximum monthly mean, and second maximum monthly mean. For example, the national average annual mean for 2003–2005 was 0.09 $\mu\text{g}/\text{m}^3$, the corresponding national maximum quarterly mean was 0.17 $\mu\text{g}/\text{m}^3$, the national maximum monthly mean was 0.31 $\mu\text{g}/\text{m}^3$, and the national second maximum monthly mean was 0.21 $\mu\text{g}/\text{m}^3$.

Similar declines were seen for other countries over this period. Table 6.10 shows non-U.S. global air lead values reported from 1997 to

TABLE 6.10 Non-U.S. Global Urban Air Lead Levels, 1997–2003

Location	Air Pb ($\mu\text{g}/\text{m}^3$)	References
Yerevan, Armenia	<0.04	Kurkjian et al. (2002)
Australia roadsides	0.40–1.00	Al-Chalabi and Hawker (1997)
Vancouver, Canada	0.05	Brewer and Belzer (2001)
Eleusis, Greece	0.11	Torfs and Van Grieken (1997)
Hong Kong roadsides	0.13–0.17	Chan et al. (2000)
Caesarea, Israel	0.004–0.44	Erel et al. (1997)
Jerusalem–Tel Aviv Freeway, Israel	0.76	Erel et al. (1997)
Jerusalem, Israel	0.02	Erel et al. (2002)
Bari, Italy	0.01	Torfs and Van Grieken (1997)
Malta	0.06	Torfs and Van Grieken (1997)
Cadiz, Spain	0.01	Torfs and Van Grieken (1997)
Gothenberg, Sweden roadsides	0.05–0.11	Sternbeck et al. (2002)
Geneva, Switzerland	0.05	Chiaradia and Cuppelin (2000)
Birmingham, U.K. roadsides	0.03	Harrison et al. (2003)

2003. Table 6.10 reports few levels of air lead higher than $0.20 \mu\text{g}/\text{m}^3$, and the latter are for roadside air lead monitoring. The highest measurements were for Australian roadsides, in the range of $0.40\text{--}1.00 \mu\text{g}/\text{m}^3$ (Al-Chalabi and Hawker, 1997). Hong Kong roadsides showed values $0.13\text{--}0.17 \mu\text{g}/\text{m}^3$ (Chan et al., 2000), with a value of 0.76 for an expressway between Jerusalem and Tel Aviv, Israel (Erel et al., 1997). Most reports recorded in Table 6.10 were for air lead levels around or below $0.10 \mu\text{g}/\text{m}^3$.

Air lead levels in the proximity of stationary sources such as primary and secondary lead smelters, battery operations, etc. will be quite elevated regardless of the co-occurrence of high lead levels in past years from dense traffic. Most major U.S. smelters had ceased operations as of this writing. There is one remaining primary lead smelter operating in the U.S., in Herculaneum, MO, and there are 15 U.S. secondary smelters (U.S. EPA, 2007b). Table 6.11 depicts representative air lead levels near U.S. stationary lead emissions sites from the 1970s to the present. The listing is skewed to earlier years because the number of these facilities has dropped, as have their emissions of lead. Nonetheless, recording air lead levels for these operations regardless of period provides a measure of the extent of atmospheric lead deposition on impacted soils and other surfaces.

Measurements of Indoor Air Pb Levels

Human exposures to air lead by direct inhalation are a combination of outside ambient and interior air lead. Yocum (1982) reported that U.S. homes and other buildings without air conditioning have indoor/outdoor air Pb ratios higher than those with air conditioning or those that are otherwise better sealed. A typical range for the former is $0.6\text{--}0.8$, while for the latter it is $0.3\text{--}0.5$. Davies et al. (1987) reported a ratio of 0.6 for U.K. dwellings with young children, a figure similar to that of Diemel et al. (1981) for tested residences in Arnhem, a Dutch community with a secondary lead smelter. The U.S. EPA, in its guidance material for its Integrated Exposure-Uptake Biokinetic (IEUBK) model for predicting lead exposures in children up to 84 months of age (U.S. EPA, 1994), employs an indoor air lead fraction of 0.3 that of outdoor atmospheric levels.

6.3.2 Lead-Based Paint

Lead in paint has long been recognized as an environmental source that provides the most serious levels of lead exposure for human risk populations and associated lead poisoning (see Chapters 7 and 8). Children are at particular risk, especially infants and toddlers. The severity of lead paint poisoning differs geographically around the globe and geographically within the United States, the country most severely impacted by production, sale, and use of

levels of air lead higher than $0.20 \mu\text{g}/\text{m}^3$, and lead monitoring. The highest measurements in the range of $0.40\text{--}1.00 \mu\text{g}/\text{m}^3$ (Al-Chalabi) at roadsides showed values $0.13\text{--}0.17 \mu\text{g}/\text{m}^3$ (value of 0.76 for an expressway between Erel et al., 1997). Most reports recorded in areas around or below $0.10 \mu\text{g}/\text{m}^3$. The proximity of stationary sources such as primary and secondary operations, etc. will be quite elevated. Reports of high lead levels in past years from dense smelters had ceased operations as of this writing. A primary lead smelter operating in the U.S., in 1995, are 15 U.S. secondary smelters (U.S. EPA, 1997). Representative air lead levels near U.S. stationary sources from the 1970s to the present. The listing is skewed toward those of these facilities has dropped, as have their monitoring. Recording air lead levels for these operations is a measure of the extent of atmospheric lead in air and on other surfaces.

Air Pb Levels

For direct inhalation are a combination of outdoor and indoor. Yocum (1982) reported that U.S. homes with air conditioning have indoor/outdoor air Pb levels. For those with air conditioning or those that are otherwise better insulated, the former is $0.6\text{--}0.8$, while for the latter it is $0.1\text{--}0.2$. Yocum reported a ratio of 0.6 for U.K. dwellings with air conditioning to that of Diemel et al. (1981) for tested residential communities with a secondary lead smelter. The material for its Integrated Exposure-Uptake model for predicting lead exposures in children up to age 6 (U.S. EPA, 1994), employs an indoor air lead fraction of 0.1.

Lead is recognized as an environmental source that provides a pathway for lead exposure for human risk populations (see Chapters 7 and 8). Children are at particular risk. The severity of lead paint poisoning is a global problem and geographically within the United States is most heavily impacted by production, sale, and use of

TABLE 6.11 Illustrative Global Air Lead Levels near Major Stationary Pb Emission Sources, 1971–2005

Source	Location	Air Pb ($\mu\text{g}/\text{m}^3$)	References
Primary lead smelter	Silver Valley, ID	10.3 (<1 mi)	Yankel et al. (1977)
		8.6 (1–1.5 mi)	
		4.9 (2.5–6 mi)	
		2.5 (6–15 mi)	
Primary Pb smelter, refinery, battery plants	Omaha, NE	5.0	McIntire and Angle (1973)
		May–November 1970 composite	
Primary Pb smelter	El Paso, TX	2.7 (4.8 km)	U.S. EPA (1977)
		2.4 (2.4 km)	
Primary Pb smelter	East Helena, MT	3.9 (0.5 mi)	U.S. EPA (1977)
Secondary Pb smelter, battery plant	Southern Ontario, Canada	3.8	Linzon et al. (1976)
		2.1	
Primary Pb smelter	Meza Valley, former Yugoslavia	24.2–38.4	U.S. EPA (1977); Fugas et al. (1973)
Secondary Pb smelter	California	1.7–4.0	Kimbrough and Suffet (1995)
Primary Pb smelter	Herculaneum, MO	2001: 1.3 ^a	U.S. EPA (2007a, Attachment B-22)
		2002: 0.4 ^a	
		2003: 0.4 ^a	
Zinc and copper smelters	Ajo, AZ	0.3 ^b	Hartwell et al. (1983)
	Anaconda, MT	0.3 ^b	
	Bartlesville, OK	0.4 ^b	
	Palmerton, PA	0.3 ^b	
Secondary Pb smelter	Arnhem, the Netherlands	0.3–0.5	Diemel et al. (1981)

^aAverage annual values for nine EPA Air Quality System (AQS) monitors, U.S. EPA (2007a, Table B-2).

^bAir Pb values closest to the smelter, reference distances differing among smelters.

lead-based paints (LBPs) in prior decades. However, it is likely no area of the globe has been fully free of lead paint risk.

Paint as a lead-bearing environmental medium differs qualitatively and quantitatively from other sources and pathways of lead for human contact.

LBP applied decades ago still exists on residential surfaces in millions of residences and other facilities occupied by young children. As such, it will continue to provide lead exposures to young children until these millions of units are rendered either lead-free or lead-safe for many years.

We characterize lead paint reservoirs in terms of decades, in contrast to lead emissions to the atmosphere where the emissions are associated with air lead changes that are on the order of hours or days in duration. A second contrasting element is that of the nature and extent of lead dispersal via various pathways. While the atmosphere (scaled as to specific areas or regions) is the common conduit for lead from emission sources, lead paint in each of the millions of U.S. residential units that contain it provides microscale risk across a macroscale of distribution.

LBP in the United States is an environmental medium broadly confined to the nation's older housing stock and its contiguous environmental compartments such as lead in building perimeter, i.e., "drip-line" soils, where lead comes mainly from exterior lead paint weathering and deposition onto contiguous surfaces. It is therefore appropriate to attempt to quantify the magnitude of the U.S. lead-paint problem by looking at various housing surveys. In particular, these are (1) the 2001 U.S. national housing survey for lead based on a representative sampling; (2) the 1997 American Housing Survey as a view of the national distribution of lead-painted housing; and (3) another national distribution survey called the Current Population Survey presented in 1999. Collectively, they provide the most reliable current picture for the U.S. lead paint problem. Of these, the 2001 National Survey of Lead and Allergens in Housing (NSLAH) is the most useful for purposes of this chapter and is a principal focus of this section (Jacobs et al., 2002; NSLAH, 2001).

Lead-containing paint as a medium for potential lead exposures for children and other risk groups is particularly complex for characterizing the nature and extent of human contact. First, lead paint's hazard as a solid surface on different residential areas and interior or exterior building components varies with the lead level (concentration of lead in paint samples) or loading (amounts of lead per unit area of measurement). These aspects were presented in the analytical measurements portion of this chapter. Surface condition, with deteriorating surfaces posing more of an overall risk to residents than surfaces in good condition at some specific instant in time, is another factor. This is not to imply that intact surfaces pose no risk. The regulatory scientific literature for LBP with regard to lead content, hazards to children's health from lead paint as a function of condition, etc., is a significant body of data. These topics are presented and discussed in more detail in a later part of this monograph. Here, these factors are included to the extent they are required to quantitatively describe the overall lead paint exposure picture for risk groups such as young children.

Both current and historic U.S. nationwide lead paint statistics are presented here for lead paint as a discrete lead source *per se*. Environmental

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U.S. nationwide lead paint statistics are pre-
s a discrete lead source *per se*. Environmental

pathways for lead from paint released to other media, such as lead paint-
derived dusts and leaded soils, are presented later. Lead paint statistics pre-
sented in such compendia as the NSLAH (2001) survey classified the various
categories of lead paint within those definitions established by the U.S. HUD
and the U.S. EPA. The U.S. HUD defines categories of lead paint characteri-
zation for this survey according to the definitions set forth in the 1999 U.S.
HUD lead-safe regulations. These regulations differ in some of their particu-
lars from companion regulations issued by the U.S. EPA in its final lead haz-
ard rule issued in 2001 (U.S. EPA, 2001). Both agencies, overall, have had
quite similar regulations for lead paint hazards as mandated by the U.S.
Congress in its 1992 "Title X" legislation.

LBP in U.S. housing and other structural stock is defined in regulatory
and statutory terms as lead paint present at a loading of 1.0 mg Pb/cm² or
higher and/or a lead concentration of 0.5% (5,000 ppm) or more. Two broad
parameters characterizing variation in the amounts of lead paint in U.S.
housing stock are region of the country and age of the housing units. These
two variables are closely related, however. Lead paint as an environmental
problem traces to the vintage of the lead-painted surfaces; the older the resi-
dential housing unit or the public building, the higher the likelihood of one
or more painted surfaces meeting the definition of LBP. Likewise, the older
the area of the country in terms of settlement history, the higher the likeli-
hood of LBP surfaces in buildings.

Table 6.12 tabulates the prevalence of LBP in U.S. housing stock as a
function of four geographic regions of the nation—Northeast, Midwest,
South, and West—and four housing age bands—pre-1940, 1940–1959,
1960–1977, and 1978–1998—within each of those four geographic regions.
The figures are estimates from the NSLAH (2001).

Table 6.12 makes it clear that the older the housing units in any of the
four major regions, the higher the fraction of units with LBP. For pre-1940
housing, the percentages of LBP units for the regions range from 71% for
units in the West to 91% in the Midwest. Corresponding figures for the
Northeast and South were 86% and 91%, respectively. Table 6.12 also shows
that the total number of units with LBP is highest in the two oldest areas of
the country, the Northeast and Midwest, with a lower count for the South
and the lowest number of LBP units found in the West.

Housing constructed in the 1978–1998 period, following the total U.S. ban
on use of lead paint for residential interiors, showed the lowest percentages of
LBP among housing units within each of the geographic areas. The persistence
of some measurable percentage in the face of the lead paint ban likely is
attributable to some combination of remaining lead paint in commercial chan-
nels after the ban and other factors. The persistence of significant percentages
of LBP surfaces in residential units built from 1960 to 1977 is noteworthy. The
fractions of housing units with LBP from this age band (%) and the corre-
sponding regions are: 39, northeast; 28, Midwest; 17, South; 22, West.

TABLE 6.12 U.S. Prevalence Rates^a of LBP in Housing^b by Region and Ages of Housing

Region	Years Built	Estimated Number of Units with LBP (000)	Estimated % of Units with LBP
Northeast	Pre-1940	5,957	86
	1940-1959	3,089	73
	1960-1977	1,478	39
	1978-1998	76	2
Regional subtotal/% LBP ^c		10,300	27
Midwest	Pre-1940	4,658	91
	1940-1959	4,785	81
	1960-1977	1,771	28
	1978-1998	533	11
Regional subtotal/% LBP ^c		11,747	31
South	Pre-1940	3,065	89
	1940-1959	3,431	54
	1960-1977	1,914	17
	1978-1998	1,197	8
Regional subtotal/% LBP ^c		9,607	25
West	Pre-1940	1,437	71
	1940-1959	2,866	69
	1960-1977	1,414	22
	1978-1998	225	4
Regional subtotal/% LBP ^c		5,942	16

Total U.S. housing units: 95,688,000; total U.S. LBP-containing units: 37,897,000; % of total with LBP: 40.

^aAdapted from NSLAH (2001).

^bAs defined by U.S. EPA (2001) and U.S. HUD (1999); Pb loading, XRF: ≥ 1.0 mg/cm², Pb concentration $\geq 0.5\%$.

^cRegional percent of total 37,897,000 U.S. LBP units.

Persistence of sizeable percentages of LBP in housing in all four regions from 1940 to 1959 is evident in Table 6.12. Percentages for this period seen in the Northeast, Midwest and West regions are generally not greatly less than those for pre-1940 housing, at 73%, 81%, and 69%, respectively.

Rates^a of LBP in Housing^b by Region and

Years Built	Estimated Number of Units with LBP (000)	Estimated % of Units with LBP
Pre-1940	5,957	86
1940-1959	3,089	73
1960-1977	1,478	39
1978-1998	76	2
	10,300	27
Pre-1940	4,658	91
1940-1959	4,785	81
1960-1977	1,771	28
1978-1998	533	11
	11,747	31
Pre-1940	3,065	89
1940-1959	3,431	54
1960-1977	1,914	17
1978-1998	1,197	8
	9,607	25
Pre-1940	1,437	71
1940-1959	2,866	69
1960-1977	1,414	22
1978-1998	225	4
	5,942	16

Total U.S. LBP-containing units: 37,897,000; % of total with

U.S. HUD (1999); Pb loading, XRF: $\geq 1.0 \text{ mg/cm}^2$,

U.S. LBP units.

percentages of LBP in housing in all four regions in Table 6.12. Percentages for this period seen in the East and West regions are generally not greatly less than those in the Midwest and South, at 73%, 81%, and 69%, respectively.

TABLE 6.13 U.S. Prevalence Rates^a of LBP^b from Housing with Children <6 Years Old^c by Age of Housing

Housing Ages	Estimated Number of Units (000) with LBP	Estimated % with LBP Units
Pre-1940	2,253	94
1940-1959	1,997	65
1960-1977	876	17
1978-1998	202	3

Total residential units with young children: 16,402,000; total LBP-containing units with young children: 5,328,000; % with LBP: 32.

^aAdapted from NSLAH (2001).^bAs defined by U.S. EPA (2001) and U.S. HUD (1999): Pb loading, XRF $\geq 1.0 \text{ mg/cm}^2$, Pb concentration $\geq 0.5\%$.^cDefined by U.S. CDC as high-risk group.

One area of concern about the persisting presence of LBP in U.S. housing is the equally persisting potential exposure threat to residents, particularly young children. The U.S. CDC in its 1991 and 2005 Statements on childhood lead poisoning defined young children less than six years of age as that subset of the population at highest risk because of various behavioral and developmental vulnerability factors (U.S. CDC, 1991, 2005).

Table 6.13 presents estimates of the number of U.S. housing units with both LBP and one or more children <6 years of age as a function of housing age. The oldest housing, pre-1940, was estimated in the 2002 NSLAH survey to have 2,253,000 housing units with both LBP and one or more young children. This comprises 94% of all units built pre-1940. The survey estimated 1,997,000 residential units had both one or more children and LBP for the 1940-1959 period, 65% of all units in that housing age group. For the 1960-1977 and 1978-1998 periods for housing construction, the counts of units with both LBP and one or more young children were 876,000 and 202,000, respectively. The corresponding percentages with LBP were 17% and 3%, respectively.

Overall, using the survey figures, there were 16,402,000 U.S. residential units with young children, and of these, 5,328,000 or 32% had LBP.

Urbanization as a factor in the relative likelihood of U.S. housing units having LBP was presented and quantified in this survey, as summarized in Table 6.14. Urbanization referred to (1) presence in or outside an MSA as defined by the U.S. Bureau of the Census; and/or (2) presence in one of two MSA categories differing in population size as also defined by the U.S. Bureau of the Census. Those U.S. MSAs with a population of 2,000,000 or higher had 24,967,000 residential units, of which 8,963,000 or 36%

TABLE 6.14 U.S. Prevalence Rates^a of LBP^b in Housing by Extent of Urbanization^c

Degree of Urbanization	Estimated Number of Units with LBP (000)	Estimated % Units with LBP
Total number of units (000) in MSAs ≥ 2 million population 24,967	8,963	36
Total number of units (000) in MSAs ≤ 2 million population 42,782	16,250	38
Total number of units (000) not in an MSA 21,808	10,046	46

Total units in MSAs = 67,749,000; total units with LBP in MSAs = 25,213,000; % all MSA units with LBP = 37.

^aAdapted from NSLAH (2001).

^bAs defined by U.S. EPA (2001) and U.S. HUD (1999). Pb loading, XRF: ≥ 1.0 mg/cm²; Pb concentration: $\geq 0.5\%$.

^cBased on data from U.S. Bureau of the Census.

contained LBP. MSAs with $<2,000,000$ population had 42,782,000 units, of which 16,250,000 or 38% had LBP. There were a total of 21,808,000 units outside of these MSAs, of which 10,046,000 units or 46 % contained LBP. The non-MSA tally excluded units not characterized or assigned to some metropolitan grouping. Overall, level of urbanization was not as significant a variable in prevalence of LBP in housing units as other variables such as age of housing and geographic location.

In the late nineteenth and twentieth centuries in U.S. households, LBP was used for both interior and exterior surfaces. Each type of surface presented different potential risks for contact and potential human exposures. Interior LBP posed the more significant risk in terms of duration of typical contact for young children, especially infants and toddlers. However, exterior lead paints produced a broader reach for lead contact through higher deterioration rates and wider dissemination of deteriorated paint residues. For example, exterior lead paint readily weathered over time and shed fine, high-hazard particles onto contiguous soils and as exterior dusts.

Table 6.15 presents U.S. prevalence rates for LBP-containing surfaces in housing units as a function of location within the housing structures: interior surfaces, exterior surfaces, or both interior and exterior surfaces. The survey estimated that 8,609,000 U.S. units or 9% of all units with LBP had interior LBP on some surface. By contrast, the U.S. prevalence rate for exterior surfaces was more than double the interior rate, 20,260,000 or 21% of LBP units. An estimate of 9,028,000 units, or 9% of all LBP units, is recorded in this table for those units having LBP on both interior and exterior surfaces.

Rates^a of LBP^b in Housing by Extent of

	Estimated Number of Units with LBP (000)	Estimated % Units with LBP
967	8,963	36
782	16,250	38
n	10,046	46

total units with LBP in MSAs = 25,213,000; % all MSA units

U.S. HUD (1999). Pb loading, XRF: ≥ 1.0 mg/cm²;

the Census.

<2,000,000 population had 42,782,000 units, of which 10,046,000 units or 46 % contained LBP. Units not characterized or assigned to some level of urbanization was not as significant a factor in housing units as other variables such as age and location.

In the twentieth centuries in U.S. households, LBP was found on interior surfaces. Each type of surface presented different risks and potential human exposures. Interior LBP posed a risk in terms of duration of typical contact for infants and toddlers. However, exterior lead paints posed a risk of lead contact through higher deterioration rates and deteriorated paint residues. For example, exterior surfaces over time and shed fine, high-hazard particles into exterior dusts.

Prevalence rates for LBP-containing surfaces in housing by location within the housing structures: interior surfaces on both interior and exterior surfaces. The survey found that 9% of all units with LBP had interior LBP. In contrast, the U.S. prevalence rate for exterior surfaces was the interior rate, 20,260,000 or 21% of LBP units, or 9% of all LBP units, is recorded in the survey of LBP on both interior and exterior surfaces.

TABLE 6.15 U.S. Prevalence Rates^a of LBP^b in Housing by Location in the Building

LBP Location	Estimated Number of Units with LBP (000)	Estimated % with LBP
Some interior surface	8,609	9
Some exterior surface	20,260	21
Both exterior and interior surfaces	9,028	9

No LBP in building: 57,791,000; % with LBP somewhere in building: 40.

^aAdapted from NSLAH (2001).

^bAs defined by U.S. EPA (2001) and U.S. HUD (1999): Pb loading, XRF: ≥ 1.0 mg/cm²; Pb concentration $\geq 0.5\%$ total units with LBP somewhere in building: 37,897,000.

TABLE 6.16 U.S. Housing Distributions^a of Highest Paint Lead Loading on Unit Interiors as a Function of Housing Age

Maximum Loading ^b by Increasing Pb Amount (mg/cm ²)	% of Units \geq Indicated Highest Pb Loading by Housing Age			
	Pre-1940	1940-1959	1960-1977	1978-1998
≥ 0.6	83	59	21	9
≥ 1.0	79	46	16	4
≥ 1.3	72	41	12	3
≥ 4.0	60	19	6	1
≥ 10.0	38	7	2	1

^aAdapted from NSLAH (2001).

^bLoading as maximum XRF reading (mg/cm²) in the housing unit.

Comparatively, about three-quarters of all LBP units—29,288,000 units—had exterior surfaces covered with LBP with or without interior LBP.

A critical factor in the level of toxicity risk posed by LBP is the level of lead loading or lead concentration. The former is indexed in units of mg Pb/cm² on painted surfaces using XRF detection (see Section 6.1) while the latter employs content of Pb per unit mass, typically as % Pb. All other factors being equal, the magnitude of Pb exposure hazard increases with the increase in Pb loading or concentration. Table 6.16 tabulates the relationship of the U.S. prevalence rates for selected threshold maximum lead loadings as a function of housing age. In general, the older the housing,

TABLE 6.17 U.S. Housing Distributions^a of Highest Paint Lead Loadings^b on Unit Exteriors as a Function of Housing Age

Maximum Pb Loading in Paint by Increasing Pb Amount (mg/cm ²)	% of Units with Indicated Highest Pb Loadings by Housing Age			
	Pre-1940	1940–1959	1960–1977	1978–1998
≥0.6	76	64	18	7
≥1.0	72	59	13	3
≥1.3	71	56	11	3
≥4.0	56	28	6	0
≥10.0	41	10	2	0

^aAdapted from NSLAH (2001).^bLoading as a maximum XRF reading (mg/cm²) in the housing unit.

the higher the percentage of maximum Pb loadings found in housing in the age band. This relationship prevails for interior surfaces, exterior surfaces, or LBP surfaces anywhere in the residential units.

Table 6.16 shows that, in pre-1940 housing, the rates at any threshold for the maximum measured Pb loading in housing interiors are much higher than those in any other housing age group. For example, maximum threshold Pb loadings ≥1.3, 4.0, and 10.0 mg/cm² in interiors occurred in pre-1940 housing at rates of 72%, 60%, and 38%, respectively. For the 1940–1959 age band, the corresponding Pb loading rates were 41%, 19%, and 7%. Measurable lead loadings above the definition of LBP were still estimated in the most recent age of housing, 1978–1998.

Table 6.17 similarly shows that in pre-1940 housing, the rates at any threshold for the maximum measured Pb loading on housing exterior surfaces were much higher than those in any other housing age group. For example, maximum threshold Pb loadings ≥1.3, 4.0, and 10.0 mg/cm² occurred in pre-1940 housing exterior surfaces at rates of 71%, 56%, and 41%, respectively. For the 1940–1959 age band, the corresponding Pb loadings in exteriors were 56%, 28%, and 10%. Measurable lead loading above the definition of a positive LBP reading at the ≥1.3 mg Pb/cm² threshold was also estimated for exteriors in the most recent housing studied, 1978–1998.

Table 6.18 shows that the percentage rates at any loading threshold for the maximum measured Pb loading in housing with LBP anywhere in the structures were highest in the oldest pre-1940 subset of housing stock in the United States. Prevalence rates for loadings ≥1.3, 4.0, and 10.0 mg Pb/cm² pre-1940 were 84%, 73%, and 55%, respectively. Moving through younger

Distributions^a of Highest Paint Lead Loadings^b by Housing Age

% of Units with Indicated Highest Pb Loadings by Housing Age			
Pre-1940	1940–1959	1960–1977	1978–1998
64	18	7	
59	13	3	
56	11	3	
28	6	0	
10	2	0	

(mg/cm²) in the housing unit.

maximum Pb loadings found in housing in the United States. For interior surfaces, the highest prevalence is for interior surfaces, exterior surfaces, and in the residential units.

For pre-1940 housing, the rates at any threshold for lead loading in housing interiors are much higher than for other housing age groups. For example, maximum threshold for lead loading in interiors occurred in pre-1940 housing at 41%, 19%, and 7%, respectively. For the 1940–1959 age group, the rates were 38%, 19%, and 7%, respectively. For the 1960–1977 age group, the rates were 38%, 19%, and 7%, respectively. For the 1978–1998 age group, the rates were 38%, 19%, and 7%, respectively. For the definition of LBP were still estimated in 1978–1998.

As that in pre-1940 housing, the rates at any threshold for lead loading on housing exterior surfaces are much higher than those in any other housing age group. For example, maximum threshold for lead loading on exterior surfaces occurred in pre-1940 housing at 71%, 56%, and 10%, respectively. For the 1940–1959 age group, the rates were 56%, 38%, and 10%, respectively. For the 1960–1977 age group, the rates were 56%, 38%, and 10%, respectively. For the 1978–1998 age group, the rates were 56%, 38%, and 10%, respectively. Measurable lead loading above the LBP reading at the ≥ 1.3 mg Pb/cm² threshold for lead loading in the most recent housing studied.

For percentage rates at any loading threshold for lead loading in housing with LBP anywhere in the housing unit, the highest prevalence is for the oldest pre-1940 subset of housing stock in the United States. For loadings ≥ 1.3 , 4.0, and 10.0 mg Pb/cm², the rates were 55%, respectively. Moving through younger

TABLE 6.18 U.S. Housing Distributions^a of Highest Paint Lead Loading^b on Units Anywhere in Building as a Function of Housing Age

Maximum Pb Loading in Paint By Increasing Pb Amount (mg/cm ²)	Pre-1940	1940–1959	1960–1977	1978–1998
≥ 0.6	89	80	31	15
≥ 1.0	87	69	24	7
≥ 1.3	84	65	18	5
≥ 4.0	73	34	10	1
≥ 10.0	55	14	3	1

^aAdapted from NSLAH (2001).

^bLoading as maximum XRF reading in the unit, mg/cm².

housing stock, the percentage of units meeting those thresholds in Pb loadings declined significantly. A comparison of the two highest Pb loading thresholds in Table 6.18 demonstrates marked declines in percent occurrences across the four housing age groups, ranging from 55% for the highest loading pre-1940 to 1% in housing built between 1978 and 1998.

Deterioration of lead-painted surfaces in the form of peeling, chipping, abrasion, and chewing can provide added risk of lead exposure over that provided by LBP surfaces assumed to be relatively intact. Central to any discussion of the role of deterioration in defining exposure risk is the need to keep in mind that all intact and accessible LBP surfaces can readily transition to surfaces in some stage of deterioration for a variety of environmental and socioeconomic reasons.

Table 6.19 depicts the rates of deteriorated and significantly deteriorated LBP surfaces in U.S. housing units as a function of age of the housing unit. Refer to U.S. EPA (2001) and U.S. HUD (1999) for regulatory characterization of degrees of deterioration. Of 37,897,000 U.S. units with LBP, 17,425,000 or 46% had one or more deteriorated LBP surfaces while 13,635,000 or 36% had one or more significantly deteriorated LBP surfaces. Of these overall tallies, pre-1940 housing units were 9,866,000 or 26% of all U.S. LBP housing units. Similarly, pre-1940 units with significantly deteriorated LBP surfaces comprised 7,752,000 units, 20%, of U.S. LBP units. Housing units with deteriorated LBP built from 1940 through 1959 amounted to 6,510,000 or 17% of all units with LBP, while 5,190,000 or 14% of the total LBP group were estimated to have significantly deteriorated surfaces. These figures are consistent with the data showing that older housing has higher rates of deteriorated LBP surfaces and significantly deteriorated

TABLE 6.19 U.S. Prevalence Rates^a of Deteriorated and Significantly Deteriorated LBP Surfaces by Age^b of Housing Stock Versus All LBP Housing

Housing Age Band	Number of Units with Deteriorated LBP (000)	% of Total LBP Units	Number of Units with Significantly Deteriorated LBP (000)	% of Total with Significantly Deteriorated LBP
Pre-1940	9,866	26	7,752	20
1940-1959	6,510	17	5,190	14
1960-1977	910	2	610	1.6
1978-1998	139	0.4	83	0.2
Total units/% all LBP	17,425	46	13,635	36

Total number of units with LBP = 37,897,000; total number of units with deteriorated LBP = 17,425,000; total number of units with significantly deteriorated LBP = 13,635,000.

^aAdapted from NSLAH (2001, Table 4.2).

^bAs defined by U.S. EPA (2001) and U.S. HUD (1999).

surfaces. Pre-1940 housing was that block of U.S. housing stock constructed in the period with highest use and consumption of LBP for home construction and repainting. At the other extreme, units painted in 1978 or later were those built after the Federal ban on interior LBP and most uses of exterior residential LBP.

Table 6.20 presents prevalence rates for deteriorated and significantly deteriorated units as a function of location in their structures.

Distributions of national total and individual unit average surface areas with LBP are given for interiors and exteriors of U.S. housing stock in Tables 6.21 and 6.22, respectively. Table 6.21 shows the national total and individual average LBP surface areas for interiors to be 7,448 million square feet and 259 square feet, respectively. Of the total national and typical LBP individual values, the highest contributor is from the wall-floor-ceiling category. Table 6.22 shows that the total national square footage for exterior surfaces is 29,159 million square feet while the average unit exterior LBP is 996 square feet. For both categories, the "wall" portion is the principal contributor.

It is important to keep in mind that there is no direct relationship between the area of any given LBP residential unit and the relative hazards to children for lead exposures. The lead exposure hazard is more a function of likelihood of contact, where deteriorated LBP on interior window sills and

Rates^a of Deteriorated and Significantly Deteriorated LBP by Age^b of Housing Stock Versus All LBP

% of Total LBP Units	Number of Units with Significantly Deteriorated LBP (000)	% of Total with Significantly Deteriorated LBP
26	7,752	20
17	5,190	14
2	610	1.6
0.4	83	0.2
46	13,635	36

897,000; total number of units with deteriorated LBP = 13,635,000. (U.S. HUD (1999).

as that block of U.S. housing stock constructed before and consumption of LBP for home construction extreme, units painted in 1978 or later were painted on interior LBP and most uses of exterior

valence rates for deteriorated and significantly deteriorated LBP in their structures.

total and individual unit average surface areas for interiors and exteriors of U.S. housing stock in Table 6.21 shows the national total and average areas for interiors to be 7,448 million square feet respectively. Of the total national and typical LBP contributor is from the wall-floor-ceiling category, the total national square footage for exterior LBP is 259 million square feet while the average unit exterior LBP is 17 square feet. In these categories, the "wall" portion is the principal

mind that there is no direct relationship between the relative hazards to children and the relative hazards to children from lead exposure hazard is more a function of like-deteriorated LBP on interior window sills and

TABLE 6.20 Estimated Prevalence Rates^a of Deteriorated and Significantly Deteriorated^b LBP in U.S. Housing Units with LBP

Location in Building	Number of Units with Deteriorated LBP (000)	Deteriorated LBP Units as % Total Housing Units	Number of Units with Significantly Deteriorated LBP (000)	Significantly Deteriorated LBP Units as % Total Housing Units
Interior surface	4,180	4	2,629	3
Exterior surface	7,009	7	3,487	4
Both interior and exterior surfaces	6,236	7	7,518	8
Total	17,425	18	13,634	14 ^c

Total housing units = 95,688,000.

^aAdapted from NSLAH (2001, Table 4.3).

^bAs defined by U.S. EPA (2001) and U.S. HUD (1999).

^cRounding.

TABLE 6.21 National and Single-Unit Amounts of LBP Surface Areas (ft²) for Interior Components^{a,b}

Component	National LBP Square Footage (000,000)	Average LBP Area (ft ²)/Housing Unit
Wall, floor, ceiling	4,993	173
Window	687	24
Door	911	32
Trim	499	17
Cabinets, beams, chimney	388	13
Total	7,448	259

^aAdapted from NSLAH (2001).

^bLBP as defined by U.S. EPA (2001) and U.S. HUD (1999).

troughs (wells) may pose more immediate exposure threats, as discussed in a later chapter, than LBP-covered ceilings with evidence of peeling or chipping paint. This is not at all to say that peeling and chipping LBP from poor-condition ceilings and walls pose little health hazard.

TABLE 6.22 National and Single-Unit Average LBP Surface Area (ft²) for Exterior Components^{a,b}

Component	National LBP Square Footage (000,000)	Average Unit LBP Surface Area
Wall	26,706	912
Window	365	12
Door	446	15
Trim	556	19
Porch	1,086	37
Total	29,159	996

^aAdapted from NSLAH (2001).^bLBP as defined by U.S. EPA (2001) and U.S. HUD (1999).

6.3.3 Lead in Interior and Exterior Dusts

Dusts are technically defined as solids consisting of small particulate materials that in turn derive from a variety of materials through diverse processes of deterioration. While dusts can consist solid particulates of any size, those which both contain lead and arise from environmental transformations of lead sources are generally considered to have an average diameter of 50 μm .

Interior and exterior lead-containing dusts, especially the former, are known to be one of the most important, if not the most important, pathways by which lead from original sources, in ambient air and/or in exterior or interior paints, enters human environments and then enters the bodies of exposed populations, notably young children. Pathways for generation of dust lead can occur directly from these sources or can occur indirectly. For example, interior dust lead levels or loadings can occur via deposition of particulate lead from the atmosphere directly to interior surfaces or through initial loading onto exterior surfaces. The indirect path can involve interim Pb deposition on soils and then lead migration from soils to interiors as dusts. Another indirect pathway for interior dust lead is "take-home" or occupational secondary exposure lead, where workplace leaded dusts in a leaded work environment adhere to workers' clothing, shoes, etc. and are carried home in the absence of washing or clothes-changing facilities at work.

For purposes of categorizing environmental mobility, one can subdivide the topic of dust lead by origin: exterior leaded dusts from atmospheric deposition and/or weathering lead paints and interior dusts from atmospheric lead deposition and/or leaded paints. Atmospheric lead deposition to interior or

Average Unit Average LBP Surface Area (ft²) for

Stational LBP Square Footage (000,000)	Average Unit LBP Surface Area
706	912
365	12
446	15
556	19
086	37
159	996

and U.S. HUD (1999).

Exterior Dusts

as solids consisting of small particulate materi-
variety of materials through diverse processes
can consist solid particulates of any size, those
arise from environmental transformations of
sidered to have an average diameter of 50 μm .
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important, if not the most important, pathways
sources, in ambient air and/or in exterior or inter-
environments and then enters the bodies of exposed
children. Pathways for generation of dust lead
sources or can occur indirectly. For example,
loadings can occur via deposition of particulate
ctly to interior surfaces or through initial load-
e indirect path can involve interim Pb deposi-
gration from soils to interiors as dusts. Another
dust lead is "take-home" or occupational sec-
workplace leaded dusts in a leaded work envi-
rothing, shoes, etc. and are carried home in the
-changing facilities at work.

ing environmental mobility, one can subdivide
a: exterior leaded dusts from atmospheric depo-
sitions and interior dusts from atmospheric lead
ts. Atmospheric lead deposition to interior or

exterior surfaces, in turn, can readily arise from either or both mobile and
stationary atmospheric lead sources.

Little in the way of quantitative information on lead content of dusts
impacting human populations appears in the literature before the late 1960s
and 1970s, for many of the same reasons that air lead data were little
reported: absence of both acceptable analytical Pb methodology and statisti-
cally standardized sampling methods. By the 1970s, measurements of dust
lead tracing to mobile sources, i.e., vehicular emissions of leaded gasoline
exhaust, or from stationary source air lead emissions like those from smel-
ters, were appearing in the scientific literature.

Lead in Exterior Dusts

Significant exterior (outside) leaded dust deposition occurred near vehicular
traffic, notably in the decades of leaded gasoline use for private and com-
mercial vehicles (U.S. EPA, 2008b). Table 6.23 tabulates dust lead measure-
ment results expressed in units of concentration for some illustrative U.S.
vehicular traffic areas recorded in the late 1960s and 1970s. Measurements
typically ranged up to about 10,000 ppm, and in the case of tunnels, up to
20,000 ppm. Table 6.23 generally depicts the high dust lead concentrations
measured at U.S. roadsides close to motor lanes in those years of high leaded
gasoline consumption, consisting of heavier fractions of auto exhaust particu-
late that settled close to the roadways soon after emission.

Table 6.24 presents U.S. roadside dust lead figures as dust lead loading
rates onto receiving surfaces from atmospheric lead deposition in this same
period of high-leaded fuel use. This table depicts lead loading results for out-
side dusts sampled in 77 U.S. Midwestern cities. Three cities had the highest
lead loading rates indexed by type of outside lead deposition: residential,
South Bend IN; commercial, Nashville, TN; industrial, Omaha, NE. Cities in
this particular survey overall showed the geometric mean of deposition rates
highest for industrial locales, intermediate for commercial sites, and lowest
for residential areas. The highest comparative finding for industrial zones is
to be expected based on contributions from both stationary and mobile lead
emission sources. Residential areas were lowest in lead deposition, showing
a clean auto traffic contribution and lower traffic densities than for commer-
cial zones.

These data for U.S. locations with elevated roadway/outside dust lead
residues in the era of high leaded gasoline consumption were largely repli-
cated for non-U.S. findings in developed areas around the world with rela-
tively large vehicular fleets and densely populated urban areas. Table 6.25
shows the case for locales in Europe, Asia, Canada, etc.

Roadway and other outside dust lead levels declined with the phasedown
and phaseout of leaded gasoline but the rate of decline has been understand-
ably slow given the long-term retention of deposited dusts in roadside soils

TABLE 6.23 Illustrative U.S. Roadside Dust Lead Concentrations ($\mu\text{g/g}$) in the Period of High Leaded Gasoline Consumption^{a,b}

U.S. Location	Road Site(s)	Dust Lead Concentration ($\mu\text{g/g}$)
Washington, DC	a. Busy road intersection	12,800
	b. Multiple sites	4000–8000
Chicago, IL	Proximity to expressway	6600
Philadelphia, PA	Proximity to expressway	3000–8000
New York City, NY	Proximity to expressway	2000 ^c
Detroit, MI	"Street dust"	966–1213 ^d
Various U.S. Cities	Highways and tunnels	10,000–20,000
Several U.S. Cities	"Street dusts"	300–18,000 ^e

^aAdapted from U.S. EPA (1977).^bFigures for early 1970s, U.S. EPA (1977).^cPinkerton et al. (1973).^dTer Haar and Aranow (1974).^eNriagu (1978).**TABLE 6.24** Illustrative U.S. Dust Lead Deposition Rates ($\text{mg/m}^2/\text{mo}$) in the Period of High Leaded Gasoline Consumption^{a,b}

Location of Highest Deposition Rate	Deposition Rate	Geometric Mean Deposition, 77 Cities, by Category
South Bend, IN	80 $\text{mg/m}^2/\text{mo}$	Residential 5.2
Nashville, TN	346 $\text{mg/m}^2/\text{mo}$	Commercial 9.8
Omaha, NE	137 $\text{mg/m}^2/\text{mo}$	Industrial 12.8

^aAdapted from Hunt et al. (1971) and U.S. EPA (1977).^bFor 77 Midwestern U.S. cities, gathered for the fourth quarter 1968.

and organic litter covers. Some illustrative values are presented in Table 6.26 for U.S., European, and Asian locales. While the dust lead levels are elevated well above background amounts, they are simultaneously well below comparative figures from past decades of high leaded gasoline use.

Roadside Dust Lead Concentrations ($\mu\text{g/g}$) in Gasoline Consumption^{a,b}

Road Site(s)	Dust Lead Concentration ($\mu\text{g/g}$)
a. Busy road intersection	12,800
b. Multiple sites	4000–8000
Proximity to expressway	6600
Proximity to expressway	3000–8000
Proximity to expressway	2000 ^c
"Street dust"	966–1213 ^d
Highways and tunnels	10,000–20,000
"Street dusts"	300–18,000 ^e

977).

Dust Lead Deposition Rates ($\text{mg/m}^2/\text{mo}$) in Gasoline Consumption^{a,b}

Deposition Rate	Geometric Mean Deposition, 77 Cities, by Category
80 $\text{mg/m}^2/\text{mo}$	Residential 5.2
346 $\text{mg/m}^2/\text{mo}$	Commercial 9.8
137 $\text{mg/m}^2/\text{mo}$	Industrial 12.8

^d U.S. EPA (1977).
^e averaged for the fourth quarter 1968.

Some illustrative values are presented in n, and Asian locales. While the dust lead levels kground amounts, they are simultaneously well om past decades of high leaded gasoline use.

TABLE 6.25 Illustrative Non-U.S., International Studies of Outside Dust Lead Levels ($\mu\text{g/g}$) in the Period of Elevated Gasoline Pb Consumption

Area(s)	Testing Sites	Dust Lead Level ($\mu\text{g/g}$)	References
Canada, Jamaica, New Zealand	Small urban areas	700–2,000	Fergusson and Ryan (1984)
Belgium	Busy roadways, street dust	500–2,500	Deroanne-Bauvin et al. (1987)
Hong Kong	Street dust	960–7,400	Lau and Wong (1982)
		130–3,900	
United Kingdom	Urban versus Rural street dusts	Urban, 970 mean	Day et al. (1975)
		Rural, 85 mean	
Lancaster, U.K.	Different test sites	Car parks (a) 46,300 mean	Harrison (1979)
		Car parks (b) 4,560 mean	
		Town center: 2,130 mean	
		Main roads: 1,890 mean	
London, U.K.	14 road areas in the city	Residential areas: 850 mean	Jensen and Laxen (1985)
		1,360–3,400	
The Netherlands	Roadways with high traffic density	Mean = 5,000	Rameau (1973)
Christchurch, New Zealand	Street dusts in selected neighborhoods	Mean = 1,160	Fergusson and Schroeder (1985)

Lead in Interior/Household Dusts

Relative to exterior leaded dusts, household and other interior dust lead reservoirs pose a higher lead exposure risk to vulnerable human populations such as young children. More so than roadside or other exterior leaded dusts, interior dusts generally reflect more lead source inputs, the relative size of

TABLE 6.26 Illustrative International Studies of Road Dust Pb Levels in Recent, Post-Gasoline Lead Use Years

Locale	Road Type	Pb Level (ppm)	References
Honolulu, HI	Three paved road areas	Means: 588	Sutherland et al. (2003)
		470	
		151	
Reno-Sparks, NV	Urban paved road	~100	Gillies et al. (1999)
San Joaquin Valley, CA	Six test areas of different road types	Means: Urban paved road, 161	Chow et al. (2003)
		Rural paved road, 57	
		Composite paved road, 109	
		Agricultural unpaved road, 58	
		Residential unpaved road, 203	
Oslo, Norway	Urban paved road	Unpaved composite road, 101	de Miguel et al. (1997)
		180	
		1,927	
Madrid, Spain	Urban paved road	1,061	de Miguel et al. (1997)
Hong Kong	Two urban paved roads	1,209	Ho et al., 2003

these inputs depending on specific circumstances. For example, old, deteriorated housing in United States' older, inner cities often have significant inputs from deteriorating leaded paints to household dusts. Interior/household dusts can also arise from factors other than purely environmental ones. Family members working in lead dust-generating industries and businesses pose the risk of secondary, indirect lead exposures of young children in the family when workplace dusts are brought home. The literature for household/interior dusts typically does not assign origin of their lead content, but some extensive databases exist for source-specific contributors including

International Studies of Road Dust Pb Levels in Use Years

Pb Level (ppm)	References
Means: 588	Sutherland et al. (2003)
470	
151	
~100	Gillies et al. (1999)
Means: Urban paved road, 161	Chow et al. (2003)
Rural paved road, 57	
Composite paved road, 109	
Agricultural unpaved road, 58	
Residential unpaved road, 203	
Unpaved composite road, 101	
180	de Miguel et al. (1997)
1,927	de Miguel et al. (1997)
1,061	Ho et al., 2003
1,209	

specific circumstances. For example, old, deteriorated, older, inner cities often have significant lead dust-generating industries and businesses direct lead exposures of young children in the area are brought home. The literature for household dusts does not assign origin of their lead content, but lists for source-specific contributors including

TABLE 6.27 Illustrative International Household/Indoor Dust Levels ($\mu\text{g/g}$) or Loadings ($\mu\text{g}/\text{m}^2$)

Location	Dust Lead Concentration ($\mu\text{g/g}$)	References
Omaha, NE	18–5,600	Angle and McIntire (1979)
Cincinnati, OH (Various housing categories)	70–16,000	Clark et al. (1985)
Lancaster, U.K.	Mean: 720 Range: 510–970	Harrison 1979
New York City (Residential sites)	610–740	Pinkerton et al. (1973)
Philadelphia, PA (Industrial neighborhoods)	930–16,000	Needleman et al. (1974)
Edinburgh, Scotland (Floor dusts)	43–13,600 Median: 308	Laxen et al. (1987)
Jersey City, NJ (Floor dusts)	1,133 (PM_{10})	Adgate et al. (1998)
Midwest United States (Pb Loadings)	5,140 $\mu\text{g}/\text{m}^2$	Clayton et al. (1999)

those concerned with the relationship of deteriorating interior (and exterior) LBP to interior dust lead health hazards. These data are included in the following tables.

Illustrative household dust lead amounts, as concentrations or loadings, reported in the United States and the United Kingdom are contained in Table 6.27. Levels by both metrics are quite high, ranging in time from the 1970s to the late 1990s. This persistence of interior dust lead content extends into later years after leaded gasoline was removed from vehicular fuel.

Table 6.28 presents some typical reports of interior dust lead levels near lead smelters and other point sources situated around the world, covering several decades. There is a considerable range of levels and relatively high mean values. In terms of source rankings, highest levels are associated with primary and secondary lead smelters, followed by residences impacted by mining and milling operations. Hartwell et al. (1983) reported median leaded dust values for lead–zinc and copper smelters of between 116 and 441 ppm, but other data in Table 6.28 cite levels of several thousand ppm or even

TABLE 6.28 Illustrative International Interior Dust Pb Levels Around Smelters and Other Point Sources

Location	Testing Area	Dust Pb Level (ppm)	References
Bartlesville, OK	Zone closest to Zn smelter	441 (median)	Hartwell et al. (1983)
Palmerton, PA	Zone closest to Zn smelter	438 (median)	Hartwell et al. (1983)
Ajo, AZ	Zone closest to copper smelter	116 (median)	Hartwell et al. (1983)
Anaconda, MT	Zone closest to copper smelter	398 (median)	Hartwell et al. (1983)
Belgium	<1 km from primary Pb smelter	2,517 (estimated from original figure)	Roels et al. (1980)
Trail, BC	Area closest to primary Pb-Zn smelter	4,676 (floor) 9,014 (window)	Hertzman et al. (1991)
		457-8,100	
Arnhem, the Netherlands	Proximity to secondary Pb smelter	Mean = 1,140 (total Pb) Mean = 1,050 (fine fraction)	Diemel et al. (1981)
Derbyshire, U.K.	a. Mining area, high soil Pb b. Low soil Pb	1,050-28,000 130-3,000	Barltrop et al. (1975)
Telluride, CO	Former mining and milling area	281	Succop et al. (1998)
Midvale, UT	Former milling and smelter site	438	Succop et al. (1998)
Butte, MT	Former mining and milling site	451	Succop et al. (1998)
Leadville, CO	Former mining, milling, and smelter site	638	Succop et al. (1998)
Pribram, Czech Republic	Proximity to Pb smelter	1,984	Rieuwerts and Farago (1996)
Shipham, U.K.	Proximity to Pb mining site	786	Thornton (1988)
Port Pirie, Australia	Proximity to Pb smelter	1,407-4,590 (PM _{2.5}) 1,693-6,799 (PM _{5.3})	Oliver et al. (1999)

(Continued)

National Interior Dust Pb Levels Around
Sources

	Dust Pb Level (ppm)	References
Zn	441 (median)	Hartwell et al. (1983)
Zn	438 (median)	Hartwell et al. (1983)
copper	116 (median)	Hartwell et al. (1983)
copper	398 (median)	Hartwell et al. (1983)
primary Pb	2,517 (estimated from original figure)	Roels et al. (1980)
primary	4,676 (floor) 9,014 (window)	Hertzman et al. (1991)
secondary	457–8,100 Mean = 1,140 (total Pb) Mean = 1,050 (fine fraction)	Diemel et al. (1981)
	1,050–28,000	Bartrop et al. (1975)
	130–3,000	
g and	281	Succop et al. (1998)
g and	438	Succop et al. (1998)
g and	451	Succop et al. (1998)
g, milling, site	638	Succop et al. (1998)
Pb smelter	1,984	Rieuwerts and Farago (1996)
Pb mining	786	Thornton (1988)
Pb smelter	1,407–4,590 (PM ₂₅₀) 1,693–6,799 (PM ₅₃)	Oliver et al. (1999)

(Continued)

TABLE 6.28 Illustrative International Interior Dust Pb Levels Around Smelters and Other Point Sources—(cont.)

Location	Testing Area	Dust Pb Level (ppm)	References
Helena, MT	Proximity of primary Pb smelters	1,598	Schilling and Bain (1988)
Kellogg, ID		0–1 mi: 36,853	
El Paso, TX (Smeltertown)	Various distances from smelter	1–2 mi: 2,726 2–3 mi: 2,234 >4 mi: 2,151	Landrigan et al. (1975)

higher. Mining and milling sites, as summarized by Succop et al. (1998), show mean values of 281–451 ppm Pb.

The 2001 U.S. national housing survey described for the case of lead paint also presented details of the relationships of interior dust lead levels with LBP. Tables 6.29–6.31 show interior dust lead data for U.S. residential units with respect to lead paint-related housing variables: dust lead loadings ($\mu\text{g}/\text{ft}^2$) versus interior surfaces at different threshold values for dust Pb (Table 6.29); interior dust lead loadings versus housing age (years of construction; Table 6.30) at different threshold values for dust Pb; and the absence or presence of dust Pb hazards with respect to absence or presence of LBP (Table 6.31). Table 6.29 shows, for uncarpeted floors, that 8,512,000 U.S. housing units, or 9% of all such units, had interior dust lead at a loading of $10 \mu\text{g}/\text{ft}^2$, while the tally and percent fraction of units $\geq 40 \mu\text{g}/\text{ft}^2$, the current U.S. HUD threshold for defining a floor lead hazard, was 2,449,000 or 3% of all U.S. housing.

Window sill troughs showed dust lead loadings above the testing detection limit for 78,936,000 units, 83% of all U.S. units. A total of 8,287,000 units or 9% of the total tally had window sill dust lead loadings at or above $250 \mu\text{g}/\text{ft}^2$, the dust hazard value for window sills. Window troughs (window wells) in 16,395,000 or 17% of U.S. housing showed surface Pb loading $\geq 800 \mu\text{g}/\text{ft}^2$, the previous Pb hazard threshold value for troughs. The current window well/trough Pb level, defined as “a clearance” rather than a “hazard” standard, is $500 \mu\text{g}/\text{ft}^2$. The clearance value is a figure abatement contractors must not exceed for satisfactory hazard reduction or control.

Table 6.30 presents the distribution of dust lead loadings on bare floors, window sills, and window troughs as a function of housing age, i.e., years when the buildings were constructed. For all dust lead loading threshold values for all three surfaces, the older the housing, the higher the number of

TABLE 6.29 Distribution of Average Loadings ($\mu\text{g}/\text{ft}^2$), All Interior Dust Lead, in U.S. Housing^{a,b}

Threshold Value, Pb-Dust ($\mu\text{g}/\text{ft}^2$)	Estimated National Tally (000)	Percent of National Tally
Uncarpeted floors		
≥ 10	8,512	9
≥ 20	4,843	5
≥ 40	2,449	3
≥ 100	966	1
Window sills		
\geq Detection limit	78,936	83
≥ 125	13,875	15
≥ 250	8,287	9
≥ 500	4,900	5
Window troughs		
\geq Detection limit	72,349	76
≥ 800	16,395	17

^aAdapted from NSLAH (2001).^bDetection limits vary with tested surface, NSLAH (2001, Ch. 5).

units in that category and thereby the higher the fractional percentage. This is especially so for the threshold values corresponding to the U.S. HUD definitions of the various surface dusts lead hazard loadings: 40, floors; 250, window sills; 800, window troughs. For uncarpeted floors, pre-1940 units accounted for 2,843,000 or 16% of dust lead measurements at or above the floor standard but 0% in units built between 1978 and 1998. The pre-1940 versus 1978–1998 categories offer particularly striking comparisons for the dust Pb hazard values for window sills and troughs.

The critical role of LBP in the distributions of dust Pb loadings on interior surfaces recorded in Tables 6.29 and 6.30 can be seen in the data depicted in Table 6.31. When LBP is present and in “good” condition as defined in the national survey, 33% of units had a dust Pb hazard as defined by the U.S. HUD (1999), but in cases of significant deterioration of the LBP present, the corresponding percentage was roughly double, 61%. The absence of LBP in the unit resulted in only 6% having a dust lead hazard of some type on some surface.

Average Loadings ($\mu\text{g}/\text{ft}^2$), All Interior Dust

Estimated National Tally (000)	Percent of National Tally
8,512	9
4,843	5
2,449	3
966	1
8,936	83
3,875	15
8,287	9
4,900	5
72,349	76
16,395	17

Source: NSLAH (2001, Ch. 5).

by the higher the fractional percentage. This and values corresponding to the U.S. HUD defined dusts lead hazard loadings: 40, floors; 250, troughs. For uncarpeted floors, pre-1940 units 1% of dust lead measurements at or above the threshold. The pre-1940 units offer particularly striking comparisons for the window sills and troughs.

The distributions of dust Pb loadings on interior surfaces are shown in Figures 6.29 and 6.30. The data in Figures 6.29 and 6.30 can be seen in the data in Table 6.30. If LBP is present and in "good" condition as defined by U.S. HUD (1999), 33% of units had a dust Pb hazard as defined by U.S. HUD (1999). In cases of significant deterioration of the LBP, the percentage was roughly double, 61%. The absence of LBP was only 6% having a dust lead hazard of some

TABLE 6.30 Distribution of Maximum Surface Dust Pb Loadings ($\mu\text{g}/\text{m}^2$) in All U.S. Housing as a Function of Construction Years^{a,b}

Threshold Value Pb-Dust ($\mu\text{g}/\text{ft}^2$)	Housing Construction Years							
	Pre-1940		1940–1959		1960–1977		1978–1998	
	Number (000)	%	Number (000)	%	Number (000)	%	Number (000)	%
A. Floor Pb loadings								
≥10	7,386	42	4,938	24	2,488	9	1,153	4
≥20	4,996	29	2,784	14	1,112	4	97	0
≥40	2,843	16	1,967	10	588	4	97	0
≥100	1,114	6	935	5	280	1	97	0
B. Window sill Pb loadings								
≥Detection limit	16,803	96	18,779	91	24,729	89	21,823	73
≥125	9,028	52	5,407	26	4,097	15	1,806	6
≥250	6,943	40	3,712	18	1,755	6	1,029	4
≥500	4,980	29	2,869	14	747	3	447	2
C. Window trough Pb loadings								
≥Detection limit	14,143	86	16,406	80	20,319	73	20,969	70
≥800	8,883	51	6,286	31	3,788	14	2,252	8

^aAdapted from NSLAH (2001).^bDetection limits vary with tested surface, NSLAH (2001, Ch. 5).

TABLE 6.31 Relationship of U.S. Housing Units^a with or without Interior Dust Lead Hazards^b as a Function of Interior LBP^c

Interior Dust Lead Hazards	No Interior LBP		LBP in Good Condition		Significant LBP	
	Number (000)	%	Number (000)	%	Number (000)	%
A. Absent	62,752	94	15,244	67	2,389	39
B. Present	4,068	6	7,508	33	3,727	61
Total units	66,820	100	22,752	100	6,116	100

^aAdapted from NSLAH (2001).^bDust lead hazard as defined by U.S. HUD (1999).^cLBP as defined by U.S. HUD (1999).

6.3.4 Lead in Soils

Natural or background levels of lead in soils are quite low and largely reflect the low levels of lead in the parent rocks giving rise to the uncontaminated soils. One exception would be elevated lead levels in those limited metalliferous soils in regions of the world with that surface mineralogy and accompanying extractive industrial activities. Background levels of lead have typically been determined by sampling done in two ways. The first is soil core testings in rural or remote areas assumed to be free of contaminating, i.e., anthropogenic, activity. The second approach consists of deeper soil core measurements and analysis of soil segments below any depth assumed to be reflective of anthropogenic activity. Both strategies require supporting data ruling out any artifactual but undetected anthropogenic contributions that result in higher lead contents than expected for the unaltered soil. Extremely low air lead values would indicate no or little atmospheric anthropogenic depositions of lead onto these soils that could artificially elevate levels over true background. Vertical strata samplings of soils would require some evidence that soil layers have not been altered through, e.g., farming, grading during property development, etc. Vertical invariance in serial deep-core soil testings would indicate the testing region is below depth-variable additions from anthropogenic activities.

Measurements of soil Pb are critically dependent on the sampling methods for data gathering. As an environmental medium, soil is quite complex in its chemical, physical, and physicochemical matrix characteristics and dependence of Pb content on distributions in the soil matrix. For example, ambient air Pb depositions on soil or depositions from lead-painted surfaces weathering, or reentrained dusts from waste sites distribute the element in soil with vertical and/or horizontal soil area variability. Air Pb typically deposits onto soils from the atmosphere in the top 2–5 cm, owing to binding of Pb by bioorganic or geological material such as vegetative litter. Soil Pb also varies with distance from point or mobile sources in direct reflection of ambient air Pb. Lead releases from weathering exterior lead paint surfaces produce depositions proportional to distances of soil areas from the building foundation or “drip line.” Increased Pb levels near the drip line may also occur to some extent from airborne Pb striking sides of buildings and fallout. However, this likelihood is ruled out when fronts and backs of structures show similar Pb levels. Anthropogenic Pb differs inversely with soil particulate size with coarse fractions of soil having lower Pb content while Pb content increases with decreased soil particle size (Young et al., 2002). This particular relationship is significant for measuring soil Pb relevant to human exposures because of the propensity for small particles to adhere to children’s hands and then be ingested.

Shacklette et al. (1971) reported an arithmetic mean of 20 $\mu\text{g/g}$ (ppm) and geometric mean of 16 ppm for 863 background soil lead samples at

of lead in soils are quite low and largely reflect parent rocks giving rise to the uncontaminated elevated lead levels in those limited metalliferous world with that surface mineralogy and accompanying activities. Background levels of lead have been sampled in two ways. The first is soil samples from areas assumed to be free of contaminating activity. The second approach consists of deeper soil samples of soil segments below any depth assumed to be free of anthropogenic activity. Both strategies require supporting data but undetected anthropogenic contributions are more than expected for the unaltered soil. This would indicate no or little atmospheric anthropogenic input to these soils that could artificially elevate lead levels. Vertical strata samplings of soils would require data that have not been altered through, e.g., farming, development, etc. Vertical invariance in serial deep samplings of the testing region is below depth-variable activities.

Results are critically dependent on the sampling method. In the environmental medium, soil is quite complex with its physicochemical matrix characteristics and distributions in the soil matrix. For example, lead in soil or depositions from lead-painted surfaces varies with distance from waste sites distribute the element in the soil area variability. Air Pb typically varies in the top 2–5 cm, owing to binding of lead with particulate material such as vegetative litter. Soil Pb levels near point or mobile sources in direct reflection of lead from weathering exterior lead paint surfaces vary with distance from the building. Increased Pb levels near the drip line may also be observed from airborne Pb striking sides of buildings and fallout. This is ruled out when fronts and backs of structures are sampled. Anthropogenic Pb differs inversely with soil particles of soil having lower Pb content while Pb levels increase with soil particle size (Young et al., 2002). This is significant for measuring soil Pb relevant to human exposure because of the propensity for small particles to adhere to children.

Freitas et al. (2004) reported an arithmetic mean of 20 $\mu\text{g/g}$ (ppm) for 863 background soil lead samples at

various U.S. sites, most of which were <30 ppm. These means were quite close to the arithmetic mean of 20 ppm reported for Canadian nonagricultural soils by McKeague and Wolynetz (1980). Rural soils in the United Kingdom were reported to have geometric means of 42 ppm (Davies, 1983) and 48 ppm (McGrath, 1986).

There is a global literature on the topic of lead contamination of soils, particularly for industrialized countries. A notable feature of Pb in soils is its persistence there, with studies showing a residence time or half-life estimated to range from decades to centuries (U.S. EPA, 2006). For example, Freitas et al. (2004) reported a mean soil Pb of 2,694 ppm for samples gathered at a long-defunct Portuguese copper mining site worked in pre-Roman and Roman times.

Retention of Pb in soils translates to concentrations of the element which do not vary widely over time, in contrast to data for atmospheric Pb emissions which show considerably more rapid changes in response to source emission rates. As one consequence, alterations in metallurgical operations such as primary lead smelters in the form of more efficient Pb emission controls lead to reductions in atmospheric air Pb and dustfall Pb but not reductions in lead content of nearby soils (Hilts, 2003).

Table 6.32 presents a range of largely urban soil Pb measurements gathered using diverse sampling methods and for various purposes from around the globe. Soil Pb content for urban properties in the United States typically range over 1,000 ppm expressed in various metrics, and in some cases, well above this figure. Central New Orleans, LA, provided measurements ranging up to 69,000 ppm. Yard soils were typically well above garden soil samples, expected from tilling soils when planting. The deeper the tilling, the greater the vertical mixing of higher Pb levels in upper strata with lower concentrations deeper in the soils. Soils less impacted by typical Pb input sources show lower values.

The impact of exterior LBP weathering over time on adjacent, i.e., drip-line and other perimeter soil, zones is apparent in Table 6.32. Ter Haar and Aranow (1974) found that soil Pb adjacent to LBP-containing wood frame buildings was much higher in Pb level than soil Pb levels 10 or 20 ft from the foundation. Urban Detroit, MI, soils showed Pb at 2,349 ppm at 2 ft away, versus a much lower 447 ppm at 10 ft. Similar differences with increasing distance were observed for rural buildings. The levels of Pb in exterior paints used in U.S. housing differed with housing age in both frequency of use and Pb concentrations or loadings used, so that the older the housing the higher the Pb content and the higher likelihood lead paint was used, especially for exterior surfaces. Subsequent outside weathering of age-variable painted surfaces, in turn, would result in differing amounts of Pb in contiguous soils. Stark et al. (1982) showed that yard soils on lots with housing built in 1920–1929 were almost 10-fold higher in Pb content than were yards with younger units, built in 1970–1977.

TABLE 6.32 Illustrative Lead Levels ($\mu\text{g/g}$, ppm) in Global Soils^{a,b,c}

Locale	Soil Sites	Pb Concentration ($\mu\text{g/g}$)	References
Various U.S. cities in a U.S. HUD program	Urban soil collections	1,043 ppm (GM) ^d	Clark et al. (2004)
Baltimore, MD, St. Paul, MN soils	Gardens or surface sampling	Baltimore, MD: Median 100 St. Paul, MN: Median 228	Chaney and Mielke (1986)
Three CA cities: Oakland, Los Angeles, Sacramento	Yard soils	Oakland: GN 897 Los Angeles: GM 188 Sacramento: GM 227	Sutton et al. (1995)
Central New Orleans, LA	Soils	Up to 69,000	Ter Haar and Aranow (1974)
Detroit, MI	Urban soils versus frame house distance	2 ft, front: 2,349 10 ft, front: 447	Ter Haar and Aranow (1974)
Rural area, MI	Soils versus frame house distance	2 ft: 2,529 10 ft: 609 20 ft: 209	Ter Haar and Aranow (1974)
Miami, FL	Urban soils	93	Chirinje et al. (2004)
Cincinnati, OH	Roadside soils	59–1,980	Turer et al. (2001)
New Haven, CT	Yard soils, variable housing ages	131, 1970–1977 1,273, 1920–1929	Stark et al. (1982)
Charleston, SC	Household soils	9–7,890, depending on traffic, LBP	Galke et al. (1975)
Chelsea, MA	Soils around a painted bridge being remediated	8,127: under bridge <30 m: 3,272 30–80 m: 457 100 m: 197	Landrigan et al. (1982)

(Continued)

Levels ($\mu\text{g/g}$, ppm) in Global Soils^{a,b,c}

	Pb Concentration ($\mu\text{g/g}$)	References
	1,043 ppm (GM) ^d	Clark et al. (2004)
for surface	Baltimore, MD: Median 100 St. Paul, MN: Median 228	Chaney and Mielke (1986)
	Oakland: GN 897	
	Los Angeles: GM 188	Sutton et al. (1995)
	Sacramento: GM 227	
	Up to 69,000	Ter Haar and Aranow (1974)
ils versus use distance	2 ft, front: 2,349 10 ft, front: 447	Ter Haar and Aranow (1974)
	2 ft: 2,529	
us frame stance	10 ft: 609 20 ft: 209	Ter Haar and Aranow (1974)
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soils	59–1,980	Turer et al. (2001)
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und a bridge being ed	8,127: under bridge <30 m: 3,272 30–80 m: 457 100 m: 197	Landrigan et al. (1982)

(Continued)

TABLE 6.32 Illustrative Lead Levels ($\mu\text{g/g}$, ppm) in Global Soils^{a,b,c} — (cont.)

Locale	Soil Sites	Pb Concentration ($\mu\text{g/g}$)	References
Omaha, NE	Urban soils	16–4,792	Angle and McIntire (1982)
Boston, MA	Urban soils in Boston area	7–13,240 Mean = 702	Rabinowitz and Bellinger (1988)
Egypt	Multiple cities urban soils	23–200	Badawy et al. (2002)
United Kingdom	Urban soils	Up to 14,100	Culbard et al. (1988)
New Zealand	Wellington urban soils near old housing	GM: 21–1,890, increasing with housing age	Bates et al. (1995)
The Netherlands cities	Urban area garden soils	GMs: 43–336	Brunekeerf et al. (1983)
Toronto, Canada	Urban soils in Toronto, Windsor	48–54	O'Heany et al. (1988)

^aMainly urban soils not affected by smelting, mining, and milling.^bReflects periods of leaded gasoline use or recent phaseout data.^cVarious soil-sampling methods used.^dGM = geometric mean.

Urban soil Pb values outside the United States show similar marked elevations over background values. Culbard et al. (1988), for example, reported U.K. urban soils ranging up to 14,100 ppm. Urban soils adjacent to LBP-containing housing in older sections of Wellington, New Zealand, were found to increase in Pb content with increasing housing age (Bates et al., 1995).

A large database has developed with reference to soil Pb values derived largely from Pb point sources, particularly smelting, milling, and mining sites. These point sources are often in rural areas, especially primary metal smelters, where extractive industrial activity will typically be the dominant contributor of Pb emissions to site and nearby community soils. By contrast, secondary Pb smelters or lead-acid battery manufacturing plants may be found in either rural or urban/suburban areas. Soil Pb contamination from extractive industrial operations can occur through several pathways: deposition of point source-generated atmospheric Pb, fugitive dusts windblown

from on-site storage to soils offsite, or by deposition of windborne Pb dusts from tailing waste piles on community yards.

One of the largest extractive industry sites in the world was the Bunker Hill lead and zinc smelting, milling, and mining complex in the Silver Valley of Idaho. This site, closed in 1981 after more than a century of operation, soon became a U.S. EPA Superfund hazardous waste site. In the early 1970s, while the complex was still operating, an extensive series of environmental contamination studies began over concerns about childhood lead poisonings within nearby communities (Walter et al., 1980; Yankel et al., 1977) and was followed by multiple environmental regulatory characterizations of the Bunker Hill complex and its contiguous communities. These characterizations included the nature and extent of the Bunker Hill Pb waste stream within and downstream of the complex, including the levels of Pb and other geochemical contaminants in soils, dusts, air, and plant life as well as exposure levels in humans and ecological populations.

Table 6.33 is a tally of soil Pb levels measured within two communities significantly impacted by the Bunker Hill operations, Kellogg and Smelterville, ID, for the period 1974–1993, a period bracketing many of the

TABLE 6.33 Average Soil Pb Levels ($\mu\text{g/g}$, ppm) Versus Calendar Years of Intervention at the Silver Valley, ID, Smelter Site^{a,b,c,d}

Year	Kellogg, ID ^e		Smelterville, ID ^e	
	AM ^f	GM ^f	AM ^f	GM ^f
1974	3,073	2,255	7,386	5,770
1975	3,918	2,658	5,581	3,907
1983	—	—	6,231	4,188
1988	3,195	2,609	2,932	2,198
1989	2,880	2,376	2,900	1,725
1990	1,572	608	1,906	719
1991	846	226	1,180	292
1992	994	276	1,264	292
1993	772	223	1,639	339

^aAdapted from TerraGraphics Environmental Engineering (2000) as prepared for Idaho Department of Health and Welfare.

^bFor the years 1974–1993.

^cSmelting operation ended in 1981.

^dDesignated a Superfund site by the EPA.

^eCommunity highly affected by Pb emissions.

^fAM, arithmetic mean; GM, geometric mean.

ffsite, or by deposition of windborne Pb dusts
community yards.

ve industry sites in the world was the Bunker
milling, and mining complex in the Silver
ed in 1981 after more than a century of opera-
Superfund hazardous waste site. In the early
still operating, an extensive series of environ-
began over concerns about childhood lead poi-
nities (Walter et al., 1980; Yankel et al., 1977)
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its contiguous communities. These characteri-
d extent of the Bunker Hill Pb waste stream
complex, including the levels of Pb and other
soils, dusts, air, and plant life as well as expo-
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Levels ($\mu\text{g/g}$, ppm) Versus Calendar Years of
ley, ID, Smelter Site^{a,b,c,d}

Kellogg, ID ^e		Smelterville, ID ^e	
GM ^f		AM ^f	GM ^f
2,255		7,386	5,770
2,658		5,581	3,907
—		6,231	4,188
2,609		2,932	2,198
2,376		2,900	1,725
608		1,906	719
226		1,180	292
276		1,264	292
223		1,639	339

mental Engineering (2000) as prepared for Idaho Department

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studies of communities within the Bunker Hill impact zone. Clearly evident
are the declines in soil Pb levels in both communities, indexed as either
arithmetic or geometric mean values. Soil Pb levels at the time of little soil
Pb remediation, 1974 and 1975, indicated lead values in the thousands of
ppm. For example, the arithmetic means for Smelterville, ID, soil Pb values
in 1974 and 1975 were 7,386 and 5,581 ppm, respectively. Associated
figures for the geometric means were 5,770 and 3,907 ppm, respectively.
The soil Pb arithmetic mean in Smelterville in 1993, well after removals of
residential soils exceeding the Pb action level were under way, was
1,666 ppm while the geometric mean was 300 ppm.

Evidence of increased Pb contamination from other extractive industry
point sources of Pb has been seen in and around neighboring communities.
Table 6.34 summarizes some illustrative examples. Leadville, CO, the site of
many decades of smelting, milling, and mining activities dating to the nine-
teenth century, had a soil Pb mean (geometric) of 1,763 ppm (Colorado
Department of Health, 1990). A large lead–zinc smelter in Trail, British
Columbia, Canada, produced group means of soil Pb of 225–1,800 ppm
(Neri et al., 1978), values not materially different from later testings in this
community with changes in emission controls (Hilts, 2003). Derbyshire,
United Kingdom, where lead mining occurred over several hundred years,
produced group means in soil Pb ranging from 420 to 13,969 ppm.

One typical feature of extractive industry primary and secondary smelters
as contaminating point sources is the inverse relationship of soil Pb levels to
distances from the emitting sources to the soil being tested. The quantitative
features of this inverse relationship are affected by height of stack, topo-
graphical features of the areas, wind patterns, and other meteorological fea-
tures such as rainfall, etc. Godin et al. (1985) examined soil Pb versus dis-
tance at several smelter sites. Liu (2003) reported the soil Pb values at
increasing distance (m) from several smelters: 313 ppm, 100 m; 217 ppm,
5,000 m; 110 ppm, 10,000 m; 57 ppm, 20,000 m; 33 ppm, 30,000 m.

The 2001 U.S. NSLAH included data for residential soil Pb levels associ-
ated with U.S. housing units characterized in various ways: number and per-
centage distributions by soil Pb thresholds, distribution by soil Pb thresholds
and housing age, and distribution by absence or presence of significantly
deteriorated exterior LBP surfaces. Collectively, these data define the rela-
tionship of lead paint to soil lead values in and around LBP residential units.
Table 6.35 indicates that 15,299,000 U.S. housing units, 16% of the total,
were associated with soil Pb levels of 200 ppm or higher. The 400 ppm or
higher soil Pb level, which for bare, play area soils defines a regulatory soil
Pb hazard level, was projected to be found at almost 10 million housing
units, 10% of all residences. Soil Pb values of 5,000 ppm or higher were pro-
jected to be found at 1,580,000 U.S. units, or 2% of all units.

The age of U.S. housing, i.e., the year of construction, has a significant
effect on the distribution of soil Pb values ranked by differing soil Pb

TABLE 6.34 Other Illustrative Soil Pb Levels in Proximity to Smelting, Milling, and Mining Point Sources^{a,b,c}

Locale	Pb Concentration ($\mu\text{g/g}$, ppm)	References
Jasper County, MO, U.S.A.	574	Murgueytio et al. (1998)
Leadville, CO, U.S.A.	1,110 ^d	Colorado Department of Health (1990)
	1,763 ^e	
Ottawa County, OK, U.S.A.	Median = 148, white children's homes	Malcoe et al. (2002)
	Median = 103, Native American children's homes	
Midvale, UT, U.S.A.	295 ^f	Bornschein et al. (1991)
Sandy, UT, U.S.A.	362 ^f	Succop et al. (1998)
Bartlesville, OK, U.S.A.	821 ^f	Hartwell et al. (1983)
Palmerton, PA, U.S.A.	331 ^g	Hartwell et al. (1983)
Anaconda, MT, U.S.A.	424 ^g	Hartwell et al. (1983)
Derbyshire, U.K.	420–13,969, group means	Barltrop et al. (1975)
Trail, BC, Canada	225–1,800, group means	Neri et al. (1978)
Trail, BC, Canada	GMs: 559–2,002, areas closest to smelter	Hertzman et al. (1991)
Arnhem, the Netherlands	240 ^f	Diemel et al. (1981)

^aUrban or rural soils affected by smelting, milling, or mining activities.^bIncludes past and present industry history.^cVarious soil-sampling methods.^dFront yard soil, geometric mean.^eFront yard soil, arithmetic mean.^fGarden soil.^gResults closest to smelter.

thresholds. In Table 6.36, for pre-1940 housing, the oldest age group, the regulatory bare soil Pb hazard level of 400 ppm or higher was encountered in 11,613,000 U.S. residences, or 67% of all U.S. housing built before 1940. The very high soil Pb threshold of 5,000 ppm or higher was seen in almost 2 million units, or 11%. By contrast, the newest housing, built in 1978–1998, showed no discernible percentage at or above 400 ppm.

e Soil Pb Levels in Proximity to Smelting, Sources^{a,b,c}

Pb Concentration ($\mu\text{g/g}$, ppm)	References
574	Murgueytio et al. (1998)
1,110 ^d	Colorado Department of Health (1990)
1,763 ^e	
Median = 148, white children's homes	
Median = 103, Native American children's homes	Malcoe et al. (2002)
295 ^f	Bornschein et al. (1991)
362 ^f	Succop et al. (1998)
821 ^f	Hartwell et al. (1983)
331 ^g	Hartwell et al. (1983)
424 ^g	Hartwell et al. (1983)
420–13,969, group means	Barltrop et al. (1975)
225–1,800, group means	Neri et al. (1978)
GMs: 559–2,002, areas closest to smelter	Hertzman et al. (1991)
240 ^f	Diemel et al. (1981)

elting, milling, or mining activities.
history.

TABLE 6.35 Distribution of Maximum Soil Pb Levels in U.S. Housing for Indicated Thresholds: Unit Counts and Percentages^{a,b,c}

Bare Soil Threshold Pb	Number (000)	% of All U.S. Units
≥ 200	15,299	16
≥ 400	9,996	10
$\geq 1,200$	6,271	7
$\geq 2,000$	3,124	3
$\geq 5,000$	1,580	2

^aAdapted from NSLAH (2001).

^bTotal U.S. units = 95,688,000.

^cSoil-sampling methods in original report.

TABLE 6.36 Maximum Soil Pb Distributions in U.S. Housing for Indicated Soil Pb Thresholds as a Function of Construction Years^{a,b,c}

Threshold soil Pb (ppm)	Years of Construction							
	Pre-1940		1940–1959		1960–1977		1978–1998	
	Number (000)	% ^d	Number (000)	% ^d	Number (000)	% ^d	Number (000)	% ^d
≥ 200	13,314	76	9,950	48	4,495	16	1,476	5
≥ 400	11,613	67	6,283	31	2,410	9	84	0
$\geq 1,200$	6,536	37	3,922	19	686	3	—	0
$\geq 2,000$	3,929	23	2,194	11	686	3	—	0
$\geq 5,000$	1,891	11	865	4	231	1	—	0

^aAdapted from NSLAH (2001).

^bBare and covered soils.

^cSampling methods described in original report.

^dPercentage of all units in the indicated age period.

or pre-1940 housing, the oldest age group, the third level of 400 ppm or higher was encountered 67%, or 67% of all U.S. housing built before 1940. The threshold of 5,000 ppm or higher was seen in 11%. By contrast, the newest housing, built in the last 20 years, had a negligible percentage at or above 400 ppm.

Table 6.37 indicates that the absence or presence of significantly deteriorated exterior LBP is a major factor in soil Pb concentration. At the 400 ppm or higher bare soil hazard level, the presence of significantly deteriorated exterior LBP increases the Pb concentration almost fourfold (30% versus 8%). The ratios are even more telling at the upper end of the soil Pb thresholds. With significant LBP deterioration, soils with Pb $\geq 5,000$ ppm are about eightfold higher than test sites without LBP.

TABLE 6.37 Bare Soil Pb Percentage Distributions in U.S. Housing for Indicated Soil Pb Thresholds in the Absence or Presence of Significant Exterior LBP Deterioration^{a,b,c}

Threshold Bare Soil Pb (ppm)	Percent Versus Significant Exterior LBP Deterioration Status	
	Not Deteriorated	Deteriorated
≥200	13	39
≥400	8	30
≥1,200	4	24
≥2,000	2	13
≥5,000	1	8

^aAdapted from NSLAH (2001).

^bAll samplings.

^cSampling methods described in original report.

6.3.5 Lead in Human Diets

Naturally occurring levels of Pb in diets consumed by early peoples have not been extensively studied. Settle and Patterson (1980), using careful laboratory techniques, reported that fresh albacore muscle in prehistoric times was one-tenth that of modern catches. Anchovies, similarly, were enriched in Pb content by 10-fold, from 2.1 to 21 ppb. Patterson (1982) found that edible plant Pb content averaged up to 2.0 ppb in prehistoric times.

Lead in the diet has long been known to be a significant source of ingested lead in human populations, with records of lead ingestion at toxic levels tracing back to at least the Greco-Roman and Roman eras (Nriagu, 1983a,b, 1985 and other citations, Ch. 2). The intervening centuries have recorded considerable evidence of dietary lead exposures. The very early record does not distinguish vulnerability to effects of dietary lead across subsets of human populations, but by the late nineteenth century episodes of childhood poisonings from lead ingestion were recorded. Stewart (1887, 1895) reported the poisoning of children in Philadelphia, PA, from bakery product coloring adulterated with lead chromate and possibly mixed with other lead compounds, producing yellow and highly toxic icings. A number of children died.

Dietary lead differs from other media-specific lead sources and pathways for human populations in a number of ways. Lead in soils and dusts typically affects young children more than adults, while food Pb can produce more Pb intakes and exposures in adults or, equally, children and adults. Dietary

Percentage Distributions in U.S. Housing for
in the Absence or Presence of Significant
LBP Deterioration Status

Percent Versus Significant Exterior LBP Deterioration Status	
Not Deteriorated	Deteriorated
13	39
8	30
4	24
2	13
1	8

nal report.

ts

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number of ways. Lead in soils and dusts typically
han adults, while food Pb can produce more Pb
ults or, equally, children and adults. Dietary

components acquire Pb via various, complex mechanisms. For example, lea-
fy vegetables can acquire Pb by foliar deposition of airborne lead or by
growing in contaminated soils. Canned foods are further contaminated
through processing methods and lead-seamed cans, a common food container
until relatively recently. Lead can bind to foods cooked in Pb-contaminated
cooking water through Pb transfer from water.

Food components differ greatly in lead content, reflecting in part differ-
ences in production and distribution. Food Pb intakes differ as a function of,
e.g., food consumption habits. This has required attention not only to concen-
trations of lead in classes of dietary components but also to quantities of
these components consumed on a typical or daily basis. Various national or
international surveys, for that reason, have determined and reported both
levels of Pb in diet categories and population consumption patterns to permit
quantification of net exposures to lead in diet.

Some reports on dietary Pb measure lead content in diet distinct from any
Pb intakes in water used for preparation of beverages or food. This chapter
discusses drinking water Pb separately, and attempts to address the additive
nature of water Pb intakes from beverages and prepared foods when such
data can be separately identified.

National food supplies in industrialized or developed societies are often
centralized, so that national food Pb surveys in, e.g., the United States and/or
Europe, provide relevant information about typical exposures across regions
and demographic subsets of the population. There are idiosyncratic excep-
tions to this rule, however, in the case of ethnic foods such as canned goods
in lead-seamed containers imported for traditional local or selective
consumption.

The nature and extent of Pb intakes from diet are quite dependent on
human host factors of age, gender, socioeconomic status, lifestyle practices,
etc. Infants consuming infant formula reconstituted from tap water differ in
their Pb intakes from older children consuming baby foods, and the latter dif-
fer from still older children consuming foods in patterns resembling those of
adults.

Dietary Pb content is affected by contamination of dietary components on
a much different time scale than either ambient air Pb at one temporal
extreme (shortest) or soil and roadside dust Pb on the other. Food grown in
areas with air Pb deposition on those crops will reflect deposition rates from
air across the entire growing season, regardless of whether atmospheric emis-
sions vary markedly over that interval. Root crops will sustain some uptake
through the root system and by root surface contamination. Canned goods
with an acidic matrix and stored in lead-seamed cans may have lead leaching
over time of storage before use.

Lead in the diet comes from dietary components whose concentrations of
the element are relatively low, compared to levels of the substance in, say,
dusts and soils. However, unlike these other environmental media, intakes of

Pb in diet on a daily or other time scale entail much larger amounts in terms of relative mass. For example, adults typically will consume only small amounts of lead in dusts and soils, 25–150 mg for various age bands, while ingesting food at a daily rate of 1 kg or more. Consequently, total daily intake of 100 mg of dust having a concentration of 1,000 ppm Pb yields the same Pb intake as 0.1 ppm Pb in 1.0 kg of diet. Other factors differentiating diet Pb from Pb in other media can potentially include a higher relative bioavailability of food Pb once ingested.

Pb in dietary components at low concentrations versus Pb in other media dictates the need for quite sensitive measurement and sampling methods, methods that only became available for routine use in the mid–late 1970s. One should therefore be cautious in evaluating earlier Pb in human diets measurement data.

It can generally be said that, at least for food supplies consumed in developed or industrialized nations, dietary lead has declined over the past several decades, owing to declines in Pb released to food components, e.g., major reductions in lead-seamed can use to virtually zero, and reduced ambient air Pb levels. For example, evaporated milk in lead-seamed cans was heavily contaminated by Pb leaching from the seams, with Mitchell and Aldous (1974) reporting an average evaporated milk value of 202 $\mu\text{g/l}$ and a range up to 820 $\mu\text{g/l}$. This section presents data for several blocks of time, from the 1960s–1970s to the present. This is done for the same reason a longer temporal reach was used for air and other media Pb emissions over time. Body Pb accumulations in older segments of human populations reflect Pb intakes and bone Pb deposition in past years. These reservoirs of body Pb can be sources of toxic exposures through Pb resorption to blood.

National and International Dietary Pb Surveys

A number of dietary Pb surveys have been carried out in the United States and around the world. Summaries of these surveys are presented in this section. Some surveys have simply reported descriptive statistics for lead concentrations in dietary groups and levels of Pb in individual food components within those groups, e.g., measured Pb levels in cereals as a group. Other surveys have reported Pb levels in dietary components and coupled these with consumption patterns to provide intakes of food Pb in some time frame, typically as daily total intakes. Some survey reports have mainly concerned themselves with total dietary Pb intakes. This chapter confines itself to Pb levels in foods and presents intakes and uptakes in the context of human exposures in later sections.

Early attempts at quantifying Pb in human diet were plagued by absence of appropriate statistical survey methods, limited reach of any survey, and the problematic nature of measurement methods in terms of laboratory sensitivity and specificity, clean-laboratory techniques, etc. The question of

er time scale entail much larger amounts in terms of lead. For example, adults typically will consume only small amounts of lead from soils, 25–150 mg for various age bands, while the average daily intake of 1 kg or more. Consequently, total daily lead intake from a concentration of 1,000 ppm Pb yields the same amount of Pb in 1.0 kg of diet. Other factors differentiating media can potentially include a higher relative bioavailability of lead ingested.

Lead at low concentrations versus Pb in other media requires a sensitive measurement and sampling methods, which were not available for routine use in the mid–late 1970s. Caution is warranted in evaluating earlier Pb in human diets.

That, at least for food supplies consumed in developed countries, dietary lead has declined over the past several decades. In Pb released to food components, e.g., major sources of lead use to virtually zero, and reduced ambient air lead in evaporated milk in lead-seamed cans was heavily reduced. Lead coming from the seams, with Mitchell and Aldous (1977) reporting an evaporated milk value of 202 µg/l and a range of 100–400 µg/l. This presents data for several blocks of time, from the 1950s to the 1970s. This is done for the same reason a longer term trend in lead and other media Pb emissions over time. Body burden measurements of human populations reflect Pb intakes over the past years. These reservoirs of body Pb can be evaluated through Pb resorption to blood.

National Dietary Pb Surveys

Lead surveys have been carried out in the United States. Summaries of these surveys are presented in this section. Simply reported descriptive statistics for lead concentrations and levels of Pb in individual food components are presented. Measured Pb levels in cereals as a group. Other levels in dietary components and coupled these to provide intakes of food Pb in some time frame. Some survey reports have mainly concerned dietary Pb intakes. This chapter confines itself to Pb intakes and uptakes in the context of human

Lead exposure in human diet were plagued by absence of sensitive survey methods, limited reach of any survey, and lack of sensitive measurement methods in terms of laboratory sensitive laboratory techniques, etc. The question of

sensitivity is a critical one, given that even in contaminated environments dietary Pb occurs at much lower levels than in dusts and soils. The 1961 study by Schroeder and Balassa of U.S. unprocessed food components found Pb content of 0–1.3 ppm, vegetables; 0–1.4 ppm, grains; 0–3.7 ppm for meats and eggs; 0.2–2.5 ppm, seafoods; 0–1.5 ppm, condiments.

The U.S. Food and Drug Administration (U.S. FDA) has been doing surveys of Pb and other contaminants in the U.S. food supply since the 1970s. The structures of these surveys and their results have varied over the years, with combinations of food-specific Pb content, Pb content of food groups, and population subset-specific dietary Pb consumption rates. The U.S. FDA Compliance Program Evaluation for Fiscal Year 1974 (summarized in U.S. EPA, 1977) included the Heavy Metals in Food Survey. Throughout the U.S. FDA survey efforts, special focus has been on dietary lead ingestion rates in infants and toddlers, as well as older children. Besides greater toxicological vulnerability, the very young consume food at an age- and weight-specific rate that is two- to threefold higher than the rates for adults.

This early U.S. FDA survey offers some general conclusions. Most notably, canned foods typically had much higher Pb content than fresh food groups, all other Pb-contributing factors being more or less equal. For example, with adult food consumptions, the average Pb content for canned foods, 0.38 ppm, was more than twice that for noncanned samples, 0.16 ppm. Infant food lead contents included 0.33 ppm for juices and 0.09 ppm for food in jars.

Several food Pb surveys were carried out in the 1970s and 1980s. The most comprehensive U.S. survey was done by the U.S. FDA using various blocks of data, including groupings of U.S. foods for total diet profiles and Pb contents of diet components within these groupings (Beloian 1985; Pennington 1983; U.S. EPA, 1986a; U.S. FDA, 1985).

Table 6.38 lists illustrative food Pb concentrations that equal or exceed 0.05 ppm Pb fresh weight for food groups and individual foods within the groups for the U.S. population via market-basket surveys and across eight age/sex groups. Items were drawn from the total diet list of Pennington (1983) and presented in U.S. EPA (1986a), and U.S. FDA (1985). Overall, food Pb content at that time was significantly below 1.00 ppm and most items are <0.100 ppm. Canned foods were the major category of higher Pb-content foods. Of the canned products, the more acidic food items were higher overall. Comparing canned tomatoes, tomato sauce, and sauerkraut with other canned fruit and vegetables, the Pb levels were one or more orders of magnitude above natural values and represented increments of Pb contamination in the U.S. food supply during growing, processing, and marketing. Notable sources for this contamination were airborne Pb deposition and lead-seamed cans.

In the early 1980s, a second source of food Pb survey data was the 1982 Nutrition Foundation Report on the safety of Pb and Pb salts. A portion of that report tabulated U.S. and Canadian food supply lead levels. Table 6.39

TABLE 6.38 Lead Content of U.S. Dietary Items Having Pb Levels ≥ 0.05 $\mu\text{g/g}$, 1970s–Early 1980s^{a,b,c}

Category Number	Food Item	Mean ($\mu\text{g/g}$)
8	Evaporated milk	0.083
27	Beef liver	0.083
32	Canned tuna	0.159
39	Canned pork and beans	0.130
45	Canned green peas	0.136
55	Canned corn	0.111
56	Canned creamed corn	0.102
57	White dinner rolls	0.084
82	Canned peaches	0.223
84	Canned applesauce	0.094
87	Canned fruit cocktail	0.221
93	Canned pineapple	0.093
101	Canned grape juice	0.053
104	Canned orange juice	0.053
106	Canned spinach	0.649
108	Frozen collards	0.074
112	Canned sauerkraut	0.524
118	Canned tomato juice	0.084
119	Canned tomato sauce	0.258
120	Canned tomatoes	0.218
122	Canned green beans	0.099
129	Canned mixed vegetables	0.081
130	Canned mushrooms	0.255
131	Canned beets	0.103
142	Spaghetti with meat sauce	0.136
145	Chili, beef, and beans	0.102
148	Meat loaf	0.093
150	Lasagna	0.070

(Continued)

nt of U.S. Dietary Items Having Pb Levels
1980s^{a,b,c}

Food Item	Mean ($\mu\text{g/g}$)
Evaporated milk	0.083
Beef liver	0.083
Canned tuna	0.159
Canned pork and beans	0.130
Canned green peas	0.136
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Canned creamed corn	0.102
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Canned peaches	0.223
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Canned fruit cocktail	0.221
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Canned orange juice	0.053
Canned spinach	0.649
Frozen collards	0.074
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Canned tomato juice	0.084
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Canned tomatoes	0.218
Canned green beans	0.099
Canned mixed vegetables	0.081
Canned mushrooms	0.255
Canned beets	0.103
Spaghetti with meat sauce	0.136
Chili, beef, and beans	0.102
Meat loaf	0.093
Lasagna	0.070

(Continued)

TABLE 6.38 Lead Content of U.S. Dietary Items Having Pb Levels
 $\geq 0.05 \mu\text{g/g}$, 1970s–Early 1980s^{a,b,c}—(cont.)

Category Number	Food Item	Mean ($\mu\text{g/g}$)
153	Pork chow mein	0.076
157	Canned vegetable soup	0.073
176	Ice cream sandwich	0.058
187	Milk chocolate candy	0.073
189	Chocolate powder	0.055

^aAdapted from U.S. EPA (1986a, Ch. 7, Appendix D); U.S. FDA (1985).^bMeans across four market-basket surveys, 1970s–early 1980s, and across eight age/sex groups.^cTotal diet food list of Pennington (1983).

depicts the Pb content data for illustrative U.S. foods within 12 food groups, including sugar items, desserts, and various types of beverages. Lead levels were recorded for uncanned foods and canned foods where indicated. In the baby foods group (canned), evaporated milk at 0.05–0.06 ppm Pb and canned juices and drinks ranked highest. Uncanned baby juices and drinks had Pb levels about half that of canned product. The highest food Pb value in the dairy group not in cans was butter at 0.07 ppm, while canned milk was highest overall, at 0.10–0.13 ppm.

The meat/poultry/fish group had highest Pb levels overall, in terms of fresh and canned forms. Canned foods Pb in this group was in the range 0.21–1.50 ppm. Cereals, potatoes, and leafy vegetables were intermediate in lead content and canned foods in these groups were two- to threefold higher than fresh/uncanned items. Root vegetables such as carrots and beets were relatively high in Pb, canned forms being 6- to 10-fold higher than uncanned examples.

Items in the garden vegetables, fruits, oils/fats, sugar items and desserts, and beverages generally were relatively lower in Pb content. Again, canned forms of these food groups were higher in Pb than uncanned examples, up to 10-fold higher in some cases.

Table 6.40 illustrates those Pb values in U.S. food items recorded from several sources during the 1990s, limited to reported concentrations at or above indicated Pb concentrations. The data from Dudka and Miller (1999) for foods ≥ 0.03 ppm are presented as concentrations per food mass, while the FDA Total Diet Survey records Pb content $\geq 2.0 \mu\text{g}/\text{individual serving}$. Root and leafy vegetables were significantly elevated compared to other items in the Dudka and Miller report while the FDA survey noted, as was the case in

TABLE 6.39 Lead Levels in Selected U.S. Dietary Items from the 1982 Nutrition Foundation Report on Safety of Lead and Lead Salts in Food^{a,b,c}

Food Group	Food Item	Lead Levels ($\mu\text{g/g}$, ppm)	
		Uncanned	Canned
Baby foods	Milk substitute	0.02	
	Mother's milk	0.03	
	Evaporated milk		0.05–0.06
	Meat/meat dinners	0.03–0.04	
	Vegetables	0.04	
	Juices and drinks	0.03	0.06
Dairy products	Milk	0.02	0.10–0.13
	Cheese	0.05	
	Butter	0.07	
Meat, poultry, fish	Beef, pork, lamb-veal	0.06	0.24
	Poultry	0.12	0.24
	Fish, excludes sardines	0.04–0.08	0.21–0.51
	Sardines	0.65	1.5
	Eggs	0.17	
	Cold cuts	0.06	0.24
Cereals	Breads	0.08	
	Flours	0.05	
	Cakes, cookies	0.03–0.05	
	Hot cereals	0.02	
	Ready-to-eat cereals	0.11	
	Rice, pasta	0.06	0.11
Potatoes	Corn	0.01	
	White cooked	0.05	0.12
	Sweet potatoes	0.08	0.25

(Continued)

Selected U.S. Dietary Items from the 1982 t on Safety of Lead and Lead Salts

Item	Lead Levels ($\mu\text{g/g}$, ppm)	
	Uncanned	Canned
Substitute	0.02	
er's milk	0.03	
rated milk		0.05–0.06
meat dinners	0.03–0.04	
ables	0.04	
and drinks	0.03	0.06
	0.02	0.10–0.13
e	0.05	
	0.07	
pork, lamb-veal	0.06	0.24
y	0.12	0.24
excludes sardines	0.04–0.08	0.21–0.51
es	0.65	1.5
	0.17	
cuts	0.06	0.24
s	0.08	
	0.05	
y, cookies	0.03–0.05	
ereals	0.02	
-to-eat cereals	0.11	
pasta	0.06	0.11
	0.01	
cooked	0.05	0.12
potatoes	0.08	0.25

(Continued)

TABLE 6.39 Lead Levels in Selected U.S. Dietary Items from the 1982 Nutrition Foundation Report on Safety of Lead and Lead Salts in Food^{a,b,c}—(cont.)

Food Group	Food Item	Lead Levels ($\mu\text{g/g}$, ppm)	
		Uncanned	Canned
Leafy vegetables	Cabbage, broccoli	0.01–0.04	0.08
	Lettuce, other greens	0.12–0.15	0.39
	Salads	0.03–0.08	0.17
Legumes	Beans	0.01–0.04	0.16–0.22
	Peas	0.03	0.27
	Soups	0.04	0.10–0.13
Root vegetables	Carrots	0.14	0.13
	Onions	0.18	0.32
	Beets	0.01	0.10–0.11
	Parsnips, turnips	0.05	0.32
Garden vegetables	Tomatoes	0.05–0.08	0.30–0.37
	Tomato paste	0.03	0.19–0.47
	Tomato juice	0.05	0.22
	Squash, pumpkin	0.03	0.36
	Peppers	0.02	0.32
	Juices	0.01–0.02	0.12–0.13
Fruits	Citrus	0.01	0.39
	Apples	0.02	0.22
	Apple juice	0.02	0.13
	Cherries	0.02	0.39
	Cranberries	0.05	0.25
	Grapes	0.01	0.28
	Peaches	0.01–0.03	0.19–0.39
	Pears	0.02	0.18–0.19

(Continued)

TABLE 6.39 Lead Levels in Selected U.S. Dietary Items from the 1982 Nutrition Foundation Report on Safety of Lead and Lead Salts in Food^{a,b,c}—(cont.)

Food Group	Food Item	Lead Levels ($\mu\text{g/g}$, ppm)	
		Uncanned	Canned
Oils and Fats	Salad dressing	0.01–0.02	
	Cooking oils	0.02	0.10
	Nuts	0.02	0.20
Sugar, desserts, jellies, etc.	Sugar	0.03	
	Chocolate	0.08	
	Candy	0.03	
	Puddings	0.01–0.03	
	Jellies	0.01	
	Pickles, olives	0.10	0.70
Beverages	Soft drinks	0.01–0.02	0.14–0.21
	Coffee	0.01	
	Tea	0.01	0.07
	Alcoholic beverages	0.01	0.02

^aAdapted from combined U.S. surveys in Nutrition Foundation (1982) report.

^bTwelve food groups; includes beverages.

^cUncanned, plus canned levels where available.

earlier surveys, that canned foods were significantly higher in Pb content than uncanned items. Among beverages, wine was highest at 7.7 $\mu\text{g/serving}$.

The most recent year for FDA survey efforts was 2004. The raw data set had all classes of contaminants and hundreds of food items containing each of those substances. Some food items shown in earlier surveys are not listed in the 2004 compilation, while some foods in this latest survey do not appear in earlier surveys. For purposes of this chapter, FDA 2004 dietary Pb data were first isolated from the full data set. Selected food items were then tabulated. Table 6.41 provides the illustrative 2004 data, along with Pb detection and quantitation measurement limits. It is readily apparent from Table 6.41 that virtually all foods in the U.S. FDA (2004) diet survey were very low in Pb, most items presenting as not measurable, or at trace (ppb) values. This marked decline for lead content in the U.S. diet is generally consistent with the removal of lead in gasoline, the abandonment of any lead-seamed

TABLE 6.41 Lead Levels in Selected Food Items for the U.S. FDA Total Diet Survey (2004)^{a,b,c}

Food Number	Food Name	Average Concentration/ppm
1	Milk, whole fluid	TR 0.002
3	Milk, chocolate, lowfat, fluid	TR 0.002
10	Cheese, American, processed	TR 0.005
12	Cheese, cheddar, natural (sharp/mild)	0.000
13	Beef, ground, regular, pan-cooked	TR 0.006
17	Ham, cured (not canned), baked	0.000
18	Pork chop, pan-cooked with oil	TR 0.003
20	Pork bacon, oven-cooked	0.000
21	Pork roast, loin, oven-roasted	0.000
26	Turkey breast, oven-roasted	0.000
29	Bologna (beef/pork)	TR 0.005
34	Fish sticks or patty, frozen, oven-cooked	TR 0.003
35	Eggs, scrambled with oil	0.000
39	Pork and beans, canned	0.000
42	Lima beans, immature, frozen, boiled	0.000
46	Peas, green, frozen, boiled	TR 0.002
47	Peanut butter, creamy	TR 0.008
50	Rice, white, enriched, cooked	0.000
54	Corn, fresh/frozen, boiled	0.000
55	Corn, canned	TR 0.002
58	Bread, white, enriched	0.000
69	Noodles, egg, enriched, boiled	TR 0.004
71	Corn flakes cereal	TR 0.005
73	Shredded wheat cereal	TR 0.006
78	Apple (red), raw (with peel)	0.000
80	Banana, raw	0.000

(Continued)

Selected Food Items for the U.S. FDA Total

	Average Concentration/ppm
fluid	TR 0.002
late, lowfat, fluid	TR 0.002
erican, processed	TR 0.005
ddar, natural	0.000
d, regular, pan-cooked	TR 0.006
(not canned), baked	0.000
pan-cooked with oil	TR 0.003
oven-cooked	0.000
oin, oven-roasted	0.000
st, oven-roasted	0.000
ef/pork)	TR 0.005
r patty, frozen, d	TR 0.003
bled with oil	0.000
beans, canned	0.000
, immature, frozen, boiled	0.000
, frozen, boiled	TR 0.002
er, creamy	TR 0.008
, enriched, cooked	0.000
/frozen, boiled	0.000
ed	TR 0.002
te, enriched	0.000
gg, enriched, boiled	TR 0.004
cereal	TR 0.005
wheat cereal	TR 0.006
, raw (with peel)	0.000
w	0.000

(Continued)

TABLE 6.41 Lead Levels in Selected Food Items for the U.S. FDA Total Diet Survey (2004)^{a,b,c} — (cont.)

Food Number	Food Name	Average Concentration/ppm
83	Peach, raw/frozen	0.000
85	Pear, raw (with peel)	0.000
87	Fruit cocktail, canned in light syrup	TR 0.009
93	Pineapple, canned in juice	TR 0.010
98	Orange juice, frozen conc., reconstituted	0.000
99	Apple juice, bottled	TR 0.005
107	Spinach, fresh/frozen, boiled	TR 0.005
108	Collards, fresh/frozen, boiled	TR 0.005
109	Lettuce, iceberg, raw	0.000
110	Cabbage, fresh, boiled	0.000
113	Broccoli, fresh/frozen, boiled	0.000
114	Celery, raw	0.000
117	Tomato, raw	0.000
119	Tomato sauce, plain, bottled	TR 0.003
122	Green beans, canned	TR 0.003
125	Pepper, sweet, green, raw	TR 0.002
128	Onion, mature, raw	0.000
131	Beets, canned	TR 0.004
136	Potato, boiled (without peel)	0.000
137	Potato, baked (with peel)	TR 0.011
148	Meatloaf, beef, homemade	0.000
156	Soup, tomato, canned, condensed, prepared with water	0.000
164	Butter, regular (salted)	0.000
166	Mayonnaise, regular, bottled	0.000
169	Sugar, white, granulated	0.000
170	Syrup, pancake	0.000

(Continued)

TABLE 6.41 Lead Levels in Selected Food Items for the U.S. FDA Total Diet Survey (2004)^{a,b,c} — (cont.)

Food Number	Food Name	Average Concentration/ppm
187	Candy bar, milk chocolate, plain	TR 0.015
191	Carbonated beverage, cola, regular	0.000
194	Carbonated beverage, cola, low-calorie	0.000
197	Tea, from tea bag	0.000
198	Beer	0.000
199	Wine, dry table, red/white	TR 0.008

^aAccessed from U.S. FDA database online, November 22, 2010 <http://www.fda.gov/Food/FoodSafety/FoodContaminantsAdulteration/TotalDietStudy/ucm184293.htm>.

^bSelected to represent illustrative major U.S. dietary components.

^cTR = trace concentration.

TABLE 6.42 U.S. Total Diet Pb Survey for Canned Foods (μg/g): Lead Levels in Pb-Seamed versus Non-Pb-Seamed Cans^{a,b,c}

Canned Food Item	Pb Level, Lead Cans ^d	Pb Level, Nonlead Cans ^d
Tuna	0.77	0.05
Orange juice	0.08	0.02
Applesauce	0.26	0.08
Apple juice	0.10	0.02
Fruit punch	0.08	0.03
String beans	0.26	0.03
Baked beans	0.27	0.02
Tomatoes	0.27	0.03
Chicken noodle soup	0.12	N.R. ^e
Vegetable soup	0.18	0.01

^aAdapted from Capar (1990) and Bolger et al. (1991).

^bAdult canned foods also eaten by young children.

^cFor fiscal years 1982–1985.

^dRounding.

^eNot reported; Bolger et al. (1991).

Selected Food Items for the U.S. FDA Total (cont.)

Name	Average Concentration/ppm
Bar, milk chocolate, plain	TR 0.015
Sweetened beverage, cola, regular	0.000
Sweetened beverage, cola, diet	0.000
Tea bag	0.000
	0.000
Dried fruit, red/white	TR 0.008

Source: online, November 22, 2010 <http://www.fda.gov/Food/Contaminants/TotalDietStudy/ucm184293.htm>.
 Major U.S. dietary components.

Lead Pb Survey for Canned Foods ($\mu\text{g/g}$): Lead vs. Non-Pb-Seamed Cans^{a,b,c}

Pb Level, Lead Cans ^d	Pb Level, Nonlead Cans ^d
0.77	0.05
0.08	0.02
0.26	0.08
0.10	0.02
0.08	0.03
0.26	0.03
0.27	0.02
0.27	0.03
0.12	N.R. ^e
0.18	0.01

Bolger et al. (1991).
 by young children.

(1).

TABLE 6.43 Changes in Production of Lead-Soldered Food Cans (Millions) 1979–1990^{a,b,c}

Year	Number of Pb-Soldered Cans (Millions)	% Pb-Seamed Units of Total Production ^d
1979	27,576	90.29
1980	24,405	85.84
1981	20,516	74.23
1982	17,412	63.21
1983	13,891	51.56
1984	11,683	41.55
1985	8,769	31.47
1986	6,775	24.31
1987	4,282	15.09
1988	1,626	5.79
1989	1,117	3.71
1990	210	3.07

^aTabulation data provided by CMI; Communication of August 27, 1990 by R.R. Budway, CMI General Counsel, to P. Mushak.

^bOnly three-piece cans fabricated with Pb.

^cSoft drink cans not in total counting.

^d% of all two- and three-piece cans ever shipped.

The impact of lead-seamed can contributions to Pb contents of canned foods declined significantly with the phasing out of those containers. Adams (1991) showed a precipitous decline in Pb levels in canned foods from the 1982–1983 to the 1988–1989 period, the time of maximal reductions in lead-seamed can production. In FY 1983–1984, the lead-seamed container food contaminant level was only 60% that of a year earlier. Two years later, the contaminant level was down to 35%, and in 1988–1989, the level was around 10% of that found 6 years earlier.

These data are consistent with figures for production of Pb-seamed cans as a fraction of the total, tabulated in Table 6.43. These data were provided to this author by the Can Manufacturers Institute (CMI) as part of a communication from CMI General Counsel R.R. Budway to P. Mushak (August 27, 1990). In 1979, Pb-seamed food cans represented 90.3%, or 27.576 billion units of total container production. By 1984, such containers represented only 41.55%, or 11.683 billion units, of total production. The 1989 fraction was only 1.117 billion containers or 3.71% of total can production.

The corresponding count for 1990, a year later, was only one-fifth of that, 210 million containers.

A number of surveys of dietary Pb have been done for other countries. The 1982 Nutrition Foundation report used for U.S. Pb content data also reported Pb levels in the Canadian food supply. Table 6.44 has illustrative Pb levels in the Canadian food supply. Twelve food groups were sampled and individual food representatives within the groups were analyzed with respect to both uncanned and, where available, canned food levels. Canadian data showed a similar elevation in Pb content for canned versus uncanned foods as seen in U.S. analyses. Among selected dairy items, cheese had the highest Pb level, 0.24 ppm. In the meat, poultry, fish category, uncanned values were significantly elevated and levels ranged from 0.05 to 0.17 ppm. Canning increased levels up to 0.26 ppm. Uncanned cereals, potatoes, and leafy vegetables as groups were the next highest in Pb concentration, with further Pb elevation seen for canned examples. Uncanned garden fruits and vegetables were moderately elevated in Pb, but significantly elevated when canned. Beverages, overall, had the lowest Pb content across the 12 groups.

The later Canadian survey of Dabeka et al. (1987) reported lead contents of about 10 diet categories including tap water and beverages. Illustrative median Pb values of items within these categories are in Table 6.45. Five dietary categories had medians of 0.030 ppm or more: cheeses, meat/fish/poultry, starch vegetables, cereals, and miscellaneous snacks and desserts. Cheese showed the highest Pb content, ranging up to 6.78 ppm.

The most wide-ranging dietary Pb survey was the food component of the Global Environmental Monitoring System (GEMS-Food, 1991), done through the UNEP. Data were gathered from 39 nations including Canada and the United States (Galal-Gorchev, 1991). Table 6.46 sets forth 11 food categories and the mean Pb content of each. Canned foods and beverages, as expected, have the highest Pb concentrations at 0.20 ppm in each case. Fish ranked next at 0.10 ppm.

The percentage contribution of illustrative food categories in each of five nations—Australia, Canada, Finland, the Netherlands, United Kingdom—are depicted in Table 6.47. Among Canadian dietary items, vegetables and the meat/fish/poultry categories each contributed 17% of total daily Pb intake, followed by beverages and cereals/cereal products at 15% each. The Finnish diet had the highest percentages of Pb intake via cereals/cereal products (24%) and fruits (22%). Beverages comprised 20% of those diets followed closely by milk products at 17%. Dutch dietary item Pb content was highest for cereals/cereal products at 17% followed by the vegetable category at 12%. For the United Kingdom, cereals (including breads) were highest at 15%, followed by potatoes at 14%.

Chen and Gao (1993) reported results of the Chinese TDS carried out in 1990. The food consumption pattern in the form of market-basket surveys and associated contaminant levels and their intakes were determined in four

for 1990, a year later, was only one-fifth of that.

of dietary Pb have been done for other countries. A Canadian report used for U.S. Pb content data also used Canadian food supply. Table 6.44 has illustrative data on food supply. Twelve food groups were sampled and representatives within the groups were analyzed with and, where available, canned food levels. Canadian data on elevation in Pb content for canned versus uncanned foods. Among selected dairy items, cheese had the highest. In the meat, poultry, fish category, uncanned meats were elevated and levels ranged from 0.05 to 0.17 ppm. Cereals, potatoes, and vegetables were the next highest in Pb concentration, with vegetables for canned examples. Uncanned garden fruits and vegetables were elevated in Pb, but significantly elevated when canned. Potatoes had the lowest Pb content across the 12 groups. A survey of Dabeka et al. (1987) reported lead contents in foods including tap water and beverages. Illustrative data within these categories are in Table 6.45. Five food categories had medians of 0.030 ppm or more: cheeses, meat/fish/seafood, cereals, and miscellaneous snacks and desserts. The highest Pb content, ranging up to 6.78 ppm.

The 1987 dietary Pb survey was the food component of the National Monitoring System (GEMS-Food, 1991), done in 1987. Data were gathered from 39 nations including Canada (Dabeka et al., 1991). Table 6.46 sets forth 11 food categories and Pb content of each. Canned foods and beverages, as well as tap water, had Pb concentrations at 0.20 ppm in each case. Fish

and vegetables had the highest Pb content in each of five nations, Canada, Finland, the Netherlands, United Kingdom—are among Canadian dietary items, vegetables and cereals each contributed 17% of total daily Pb intake, and cereals/cereal products at 15% each. The Finnish diet had 17% of Pb intake via cereals/cereal products. Beverages comprised 20% of those diets followed by vegetables at 17%. Dutch dietary item Pb content was highest in vegetables at 17% followed by the vegetable category at 17% followed by the vegetable category at 17% followed by the vegetable category at 14%.

The reported results of the Chinese TDS carried out in 1987 showed a similar pattern in the form of market-basket surveys. Lead levels and their intakes were determined in four

TABLE 6.44 Lead Levels in Selected Canadian Dietary Items from the 1982 Nutrition Foundation Report on Safety of Lead and Lead Salts in Food^{a,b,c}

Food Group	Food Item	Lead Level (µg/g, ppm)	
		Uncanned	Canned
Dairy products	Evaporated milk	—	0.04
	Whole milk	0.02	
	Cheese	0.24	
	Butter	0.05	
Meat, poultry, fish	Beef	0.09	
	Pork	0.10	
	Veal	0.05	
	Lamb	0.05	
	Poultry	0.08	
	Eggs	0.05	
	Fish, fresh	0.05	
	Tuna		0.26
	Salmon		0.16
	Shellfish	0.05	
	Organ meats	0.10–0.17	
	Prepared meat, poultry		0.25
Cereals	Breads and rolls	0.05	
	Flour	0.08	
	Breakfast cereals, all types	0.05	
	Rice	0.05	
	Pasta	0.05	0.10
	Corn	0.03	0.36
Potatoes	Potatoes, baked and boiled	0.06	0.12
	Sweet potatoes, yams	0.08	0.25

(Continued)

TABLE 6.44 Lead Levels in Selected Canadian Dietary Items from the 1982 Nutrition Foundation Report on Safety of Lead and Lead Salts in Food^{a,b,c} — (cont.)

Food Group	Food Item	Lead Level ($\mu\text{g/g}$, ppm)	
		Uncanned	Canned
Leafy vegetables	Cabbage	0.05	
	Celery	0.03	
	Lettuce, other greens	0.07	0.13
	Asparagus	0.03	0.13
	Mushrooms	0.04	0.18
Legumes	Beans	0.03	0.16
	Peas	0.03	0.13
Root vegetables	Carrots	0.07	0.13
	Onions	0.05	
	Beets	0.05	0.17
	Turnips, parsnips	0.03	
Garden fruits	Tomatoes	0.04	0.25
	Cucumbers	0.03	
	Squash	0.03	
	Eggplant	0.03	
	Tomato juice		0.20
	Tomato paste		0.57
Fruits	Citrus	0.03	0.14
	Apples	0.14	0.17
	Apple juice		0.08
	Grapes	0.02	0.14
	Peaches	0.04	0.27
	Pears	0.04	0.22
	Pineapple	0.05	0.19
	Cherries	0.02	0.14
	Berries	0.05	0.14

(Continued)

Selected Canadian Dietary Items from the Report on Safety of Lead and Lead Salts

Food Item	Lead Level ($\mu\text{g/g}$, ppm)	
	Uncanned	Canned
Orange juice	0.05	
Carrots	0.03	
Spinach, other greens	0.07	0.13
Broccoli	0.03	0.13
Mushrooms	0.04	0.18
Beans	0.03	0.16
Peas	0.03	0.13
Potatoes	0.07	0.13
Onions	0.05	
Apples	0.05	0.17
Carrots, parsnips	0.03	
Potatoes	0.04	0.25
Peas	0.03	
Spinach	0.03	
Broccoli	0.03	
Orange juice		0.20
Orange paste		0.57
Apples	0.03	0.14
Potatoes	0.14	0.17
Orange juice		0.08
Apples	0.02	0.14
Spinach	0.04	0.27
Apples	0.04	0.22
Apple	0.05	0.19
Strawberries	0.02	0.14
Strawberries	0.05	0.14

(Continued)

TABLE 6.44 Lead Levels in Selected Canadian Dietary Items from the 1982 Nutrition Foundation Report on Safety of Lead and Lead Salts in Food^{a,b,c} — (cont.)

Food Group	Food Item	Lead Level ($\mu\text{g/g}$, ppm)	
		Uncanned	Canned
Oils and fats	Nuts	0.05	
	Cooking oils	0.02	
	Salad dressings	0.05	
Sugar and Adjuncts	Sugar	0.05	
	Syrup		0.17
	Jams and jellies	0.05	
	Puddings	0.05	
	Candy	0.06	
Drinks	Coffee, tea	0.03	
	Soft drinks		0.05
	Alcoholic beverages	0.01	

^aAdapted from Canadian Food Surveys in Nutrition Foundation (1982) report.^bTwelve food groups, includes beverages.^cUncanned levels plus canned levels where available.

major geographic groupings in China with included provinces: North 1, Heilongjiang, Liaoning, Hebei; North 2, Henan, Shaanxi, Ningxia; South 1, Shanghai, Jiangxi, Fujian; South 2, Hubei, Sichuan, Guangxi. Each regional market basket consisted of 12 food composites. For Pb, the contributions of various food groups as a national percentage of the total national Pb intake were: cereals, 39.6; vegetables, 27.5; potatoes, 7.8; meats, 5.6; fruits, 5.4; eggs, 5.1; legumes and nuts, 4.4; aquatic foods, 2.8. Milk, water, prepared beverages, and milk and sugar added relatively minor amounts of Pb to daily intake.

The various national and international surveys included food crop data for food crop components produced in uncontaminated soils. Soils in urban areas or areas impacted by lead smelters not only have elevated Pb content, as is to be expected, but crops grown there also show Pb concentrations well above samples produced in uncontaminated soils. Table 6.48 depicts food crops with high Pb content under these conditions. Finster et al. (2004) reported that leafy and root vegetables grown in urban gardens in Chicago, IL, had Pb levels well above 10 ppm. Swiss chard was found at 22–24 ppm

TABLE 6.45 Illustrative Pb Levels in Various Food Categories of the Canadian Diet^{a,b,c}

Food Category	Median (ppm)	Ranges (ppm)
Cheese (excluding cottage cheese)	0.034	0.028–6.78
Meat, fish, poultry, meat soups	0.031	0.011–0.121
Dairy products, eggs	0.003	0.001–0.082
Fruits, fruit juices, canned and fresh	0.008	0.002–0.109
Potatoes, rice, other starches	0.07	0.006–0.084
Nonstarch vegetables	0.032	0.001–0.254
Miscellaneous snacks, desserts	0.033	0.014–1.38
Cereals, bread	0.032	0.012–0.078
Beverages	0.009	<0.00005–0.029

^aAdapted from Dabeka et al. (1987) and World Health Organization (1995).^bUnits of ppm.^cCanned and uncanned items combined.**TABLE 6.46** Typical Pb Concentrations of Illustrative Foods in the GEMS-Food International Survey^{a,b,c}

Food	Pb Level (µg/g, ppm)
Cereals	0.06
Meats	0.05
Organ foods	0.02
Fish	0.10
Shellfish	0.02
Vegetables	0.05
Fruits	0.05
Roots/tubers	0.05
Eggs	0.02
Canned foods	0.20
Canned beverages	0.20

^aAdapted from Galal-Gorchev (1991).^bGEMS-Food: Food component of the Global Environmental Monitoring System, UNEP.^cData for 39 countries, including the United States.

Levels in Various Food Categories of the

	Median (ppm)	Ranges (ppm)
ese)	0.034	0.028–6.78
s	0.031	0.011–0.121
	0.003	0.001–0.082
fresh	0.008	0.002–0.109
	0.07	0.006–0.084
	0.032	0.001–0.254
	0.033	0.014–1.38
	0.032	0.012–0.078
	0.009	<0.00005–0.029

7) and World Health Organization (1995).

combined.

Concentrations of Illustrative Foods in the Survey^{a,b,c}

	Pb Level (µg/g, ppm)
	0.06
	0.05
	0.02
	0.10
	0.02
	0.05
	0.05
	0.05
	0.02
	0.20
	0.20

91).
the Global Environmental Monitoring System, UNEP.
the United States.

TABLE 6.47 Relative Contributions of Dietary Pb to Intake as a Function of Food Group Among Indicated Countries^{a,b,c}

Country	Food Group	% Total Pb Intake
Canada	Vegetables	17
	Meat/fish/poultry	17
	Beverages	15
	Cereals/products	15
	Fruits, juices	10
Finland	Cereals/products	24
	Fruits	22
	Beverages, etc.	20
	Milk/products	17
The Netherlands	Cereals/products	17
	Vegetables	12
United Kingdom	Breads/cereals	15
	Beverages	14
	Potatoes	10
Australia	Tea	20

^aAdapted from Galal-Gorchev (1991), from the GEMS-Food International Survey.

^bReported for adults.

^cFood groups contributing $\geq 10\%$ total Pb intake.

Pb, while onion samples had 21 ppm. Moseholm et al. (1992) showed lettuce grown close to a Pb smelter had a Pb level in the range of 0.7–1.3 ppm, while kale grown close to the smelter had Pb levels 0.6–2.4 ppm.

6.3.6 Lead in Drinking Water

Drinking water Pb has had a long history of producing both lead exposures and associated lead poisonings. References appeared in Greco-Roman times to the health hazards of using waters flowing in lead piping versus waters in wood pipes (see Chapter 2). This history extended into more recent decades as cities and towns appeared and grew in industrializing countries. Growth in urbanization was accompanied by growth in setting up and maintaining public drinking water systems, typically established as public utilities operated by public agencies and drawing water from both surface water and

TABLE 6.48 Selected Lead Levels of Garden Crops Produced in Contaminated Soils^{a,b,c}

Locale	Food Items	Pb Level ($\mu\text{g/g}$, ppm) ^d	References
Chicago, IL (urban gardens)	Cilantro	49	Finster et al. (2004)
	Collard greens	12	
	Coriander	39	
	Ipasote	14	
	Lemon balm	20	
	Mint	<10–60	
	Rhubarb	<10–36	
	Swiss chard	22–24	
	Carrot	10	
	Onion	21	
Copenhagen, Denmark, close to Pb smelter	Radish	12–18	Moseholm et al. (1992)
	Lettuce	0.7–1.3	
	Carrots	0.07–0.28	
	Potatoes	0.6–2.4	
	Kale	1.4–9.3	

^aAdapted from original articles.^bContamination from mobile or point sources.^cDifferent crops tested in indicated locales.^dLevels ≥ 10 ppm.

groundwater. Areas in suburban or remote locales, by contrast, often rely on private water sources such as wells.

The topic of Pb in drinking water, technologically, is a complex one in both the number of discrete steps needed to produce potable drinking water for human populations and the myriad of physical, physicochemical, and chemical factors that work to produce low, moderate, or high concentrations of Pb in drinking water sources for these populations. The environmental physical factors typically include the nature of the water chemistry, the nature of the components of water transport from sources to the residences and/or public areas such as schools for human populations, the characteristics of residential plumbing systems, and finally, patterns of water use in different risk groups in the population.

Lead Levels of Garden Crops Produced in

Food Items	Pb Level ($\mu\text{g/g}$, ppm) ^d	References
Cilantro	49	Finster et al. (2004)
Collard greens	12	
Coriander	39	
Ipasote	14	
Lemon balm	20	
Mint	<10–60	
Rhubarb	<10–36	
Swiss chard	22–24	
Carrot	10	
Onion	21	
Radish	12–18	Moseholm et al. (1992)
Lettuce	0.7–1.3	
Carrots	0.07–0.28	
Potatoes	0.6–2.4	
Kale	1.4–9.3	

point sources.
ed locales.

urban or remote locales, by contrast, often rely on wells.

Drinking water, technologically, is a complex one in the steps needed to produce potable drinking water and the myriad of physical, physicochemical, and chemical factors that can affect the water. To produce low, moderate, or high concentrations of lead from various sources for these populations. The environmental factors that include the nature of the water chemistry, the distance of water transport from sources to the residences of schools for human populations, the characteristics of the water systems, and finally, patterns of water use in different populations.

Natural levels of lead in both surface water and groundwater free of lead contamination from anthropogenic activities or arising from contamination in the course of storage, transport, and distribution to consuming populations are extremely low. A range of 0.005–10 $\mu\text{g/l}$ was estimated by NAS/NRC (1980) but the upper end of the range likely reflects artifactual contamination by Pb during laboratory testing. U.S. EPA (1986b) settled on an upper limit of 0.02 $\mu\text{g/l}$.

Untreated surface waters averaged 3.9 $\mu\text{g Pb/l}$ for 749 surface water samples, with a range of 1–55 $\mu\text{g/l}$ in the report by Durum et al. (1971). NAS/NRC (1980) reviewed surface water levels in the literature, a range from undetected to 890 $\mu\text{g/l}$, with the upper end of the range likely showing considerable contamination. U.S. EPA (1986b) selected the Patterson concentration of 0.006–0.05 $\mu\text{g/l}$ for remote streams. Proximity to sewage treatment, urban runoff, and industrial waste pollution resulted in much higher values, around 100 $\mu\text{g/l}$. Groundwater lead content has been reported in the range of 1–100 $\mu\text{g/l}$ (NAS/NRC (1980)).

A relatively high fraction of U.S. households, 84%, receive treated drinking water supplies from public or private surface water or groundwater systems. Water wells are the second largest source (U.S. EPA, 1986b). Waters leaving treatment plants in U.S. and Canadian systems are low, 2–3 $\mu\text{g/l Pb}$ (Dabeka et al., 1987; U.S. EPA, 1991).

Water Pb levels at the tap are typically much higher than concentrations of Pb in piped water leaving treatment plants (Isaac et al., 1997; Mushak and Crocetti, 1990; NAS/NRC, 1993; U.S. EPA, 1986b, 2006; WHO, 1995). Isaac et al. (1997) reported that ratios of water Pb levels to Pb concentrations measured at the tap with variable standing times ranged from 0.17 to 0.69 in water flowing into Massachusetts homes. Lead enters household and public water plumbing at a number of sites before exiting the tap. First, there are lead connectors, “pigtailed,” joining water lines to household plumbing; in the past, lengths of lead pipe were used in areas with highly corrosive soils for other forms of metal plumbing (U.S. EPA, 1985). Lead piping has largely been abandoned in favor of copper or nonmetal plumbing materials and one only finds lead plumbing in the oldest housing in older areas of countries such as the United States. Overall, the principal contributor of leachable lead in household plumbing is corrosible 50:50 lead–tin soldered plumbing joints usually found in copper plumbing. Pb-alloyed brass household water fixtures, e.g., faucets in kitchens and bathrooms, are a second significant source.

Pb leaching into household and public building plumbing from lead–tin (Pb–Sn) 50:50 solder in copper water lines has been the subject of a number of studies. When used with copper plumbing lines, Pb–Sn solder is affected by electrochemical, i.e., galvanic, reactions which produce divalent Pb ion. In general, the most significant factors affecting pipe joint solder Pb release are aggressive water, i.e., low pH, and low “hardness” or carbonate level

(U.S. EPA, 1986b), standing time in unit plumbing lines, temperature, and relative efficiency of the soldering. Other factors include age of a given type of plumbing and interior surface coating (deMora and Harrison, 1984). Subramanian et al. (1994) were able to show that any of a number of non-lead fitting solders was sufficiently resistant to galvanic leaching to be safe to use. In addition to the most widely used alternative, a 95:5 tin-antimony solder, tested materials included 94:6 tin-silver, 96:4 tin-silver, and 95.5:4.0:0.5 tin-copper-silver solders.

Quantification of Pb leach rates from simulated plumbing systems and also household plumbing systems with lead solder, copper piping, and treated or well waters have been reported. Treweek et al. (1985) used pilot-plant simulations of corrosion in domestic pipe materials to evaluate the impact of different water quality variables on releases of lead and copper from soft, low-alkalinity water consumed in Portland, OR, over a test time of 18 months. The lead-tin solder-coated copper coiling tested with chloraminated water, with ample standing time of 8 hours, showed lead leaching at the outset exceeding the then MCL of 50 $\mu\text{g/l}$, while chlorinated water only showed exceedences over the MCL for copper. Over time, the Pb leach rates declined. These findings led to a health advisory for residents to run the tap water before using to flush standing water Pb accumulations.

Thompson and Sosnin (1985) evaluated the corrosion characteristics of 50:50 Pb-Sn solder in household plumbing, using either short-term static tests in lead-soldered copper test cells (15 days) or mock-up plumbing assemblies (40 days) and three water sources differing in pH and corrosiveness. Water Pb buildup in acidic, most aggressive test, water at room temperature was initially 14 $\mu\text{g/l/hour}$ with a final rate of 11 $\mu\text{g/l/hour}$ rate. Hot water testing (60°C) for this same set of parameters gave an initial leaching rate of 48 $\mu\text{g/l/hour}$ and a final value of 55 $\mu\text{g/l/hour}$. Birden et al. (1985) studied Pb leaching from lead-soldered joints using simulated household plumbing assemblies with multiple soldered joints and several public well water sources in New England differing in relative corrosivity. One well water sample had a pH of 6.7 and low total hardness of 47 mg/l versus the second water with pH of 7.8 and hardness of 110 mg/l. The lower pH and hardness water had a Pb leaching from 50:50 tin-lead solder of 364 $\mu\text{g/l}$ compared to 80 $\mu\text{g/l}$ for the second system.

A second source of galvanic Pb leaching in domestic plumbing systems, in addition to that from lead-tin soldered joints, is household plumbing fixtures. Lead sources in fixtures include both soldered connections and the brass alloy composition with a permissible significant Pb content (3–8% Pb). Samuels and M  ranger (1984) tested lead leaching from commercially available kitchen faucets with varying details of construction in presence or absence of leaded solder in the connecting tube assemblies. Waters were tested as available from the Ottawa, Canada, water supply, in addition

ing time in unit plumbing lines, temperature, and soldering. Other factors include age of a given type of surface coating (deMora and Harrison, 1984). They were able to show that any of a number of non-ferrous alloys were sufficiently resistant to galvanic leaching to be safe. The most widely used alternative, a 95:5 tin-antimony alloy, included 94:6 tin-silver, 96:4 tin-silver, and pure silver solders.

Leach rates from simulated plumbing systems and actual systems with lead solder, copper piping, and treated water were reported. Treweek et al. (1985) used pilot-plant tests of domestic pipe materials to evaluate the impact of various variables on releases of lead and copper from soft water consumed in Portland, OR, over a test time of 8 hours. Solder-coated copper coiling tested with chloraminated water, standing time of 8 hours, showed lead leaching at a rate of 11 $\mu\text{g/l/hour}$, while chlorinated water only showed a rate of 1 $\mu\text{g/l/hour}$, the MCL for copper. Over time, the Pb leach rates increased to a health advisory for residents to run the tap water for 10 minutes to flush standing water Pb accumulations.

Birden et al. (1985) evaluated the corrosion characteristics of household plumbing, using either short-term static tests or mock-up plumbing systems. They tested three water sources differing in pH and corrosivity: distilled water, most aggressive test, water at room temperature, and water with a final rate of 11 $\mu\text{g/l/hour}$. Hot water at the same set of parameters gave an initial leaching rate of 55 $\mu\text{g/l/hour}$. Birden et al. (1985) tested lead-soldered joints using simulated household plumbing with multiple soldered joints and several public well waters differing in relative corrosivity. One well had a pH of 6.7 and low total hardness of 47 mg/l versus the other with a pH of 7.8 and hardness of 110 mg/l. The lower pH and hardness resulted in leaching from 50:50 tin-lead solder of 364 $\mu\text{g/l}$ versus 11 $\mu\text{g/l}$ in the second system.

Galvanic Pb leaching in domestic plumbing systems, particularly at lead-tin soldered joints, is household plumbing fixtures include both soldered connections and the use of pipe with a permissible significant Pb content (3–8% Pb). Birden et al. (1984) tested lead leaching from commercially available pipe with varying details of construction in presence of water in the connecting tube assemblies. Waters from the Ottawa, Canada, water supply, in addition

to a well water source and aqueous fulvic acid solution. Filtered water from those kitchen faucets with lead-soldered joints was extensive, ranging from 4 to 55 mg/l (4,000–55,000 $\mu\text{g/l}$), for an initial 24-hour stagnation (contact) period.

Several surveys of Pb in drinking water, mainly in the United States and Canada, have been carried out. These surveys, differing in their geographic and environmental scope, were done from the early 1980s to 2006. The reported water Pb levels in these surveys were illustrative of tap water statistics such as mean water Pb across cities, states, and countries and prevalence rates of water Pb exceedences for standards and guidelines as they existed at the time. For example, the U.S. EPA had a Pb in drinking water supply MCL of 50 $\mu\text{g/l}$ until 1991. In 1991, this regulatory approach changed to a two-part control protocol. First, U.S. EPA promulgated a tap water action level of 15 $\mu\text{g/l}$, not to be exceeded at the 90th percentile, for the sampled community in order to forestall some form of advisory or treatment technology change and, second, an enforceable MCL standard of 5 $\mu\text{g/l}$ (5 ppb) for water exiting public water treatment plants.

Table 6.49 presents tap water Pb levels in selected U.S., Dutch, and English water systems reported by various authors. Sampling methodology used standing, flushed, and composited samples and various types of water piping. It is clear that stagnant water had much larger amounts of Pb than flushed samples. Composite samples from random collections were intermediate in Pb concentration. Using plastic pipe resulted in relatively lower Pb content in household water, but levels were still measurable. Running, flushed water line samples depicted water system background Pb content as treated waters left the plant. The highest values in Table 6.49, not unexpectedly, were the mean of 1,075 $\mu\text{g/l}$ and the maximum figure of 2,826 $\mu\text{g/l}$ for standing water samples collected in English households having lead water lines.

More recent selected household water surveys in U.S. and Canadian households showed marked reductions in both standing and running water samples. In Table 6.50, geographic locales with soft waters and a history of aggressive plumbing corrosion showed the highest concentrations, e.g., the selected Massachusetts communities. One still saw a distinction between standing and flushed water samples, with U.S. Midwest standing levels being about fivefold higher than flushed samples.

Distribution of water Pb elevations in water systems across geography and time have been reported. Table 6.51 shows the water Pb content of U.S. urban water systems at the community-based water Pb action level of 15 $\mu\text{g/l}$ at three times: 1992, 1993, and more recently, from 1999 to 2006, with most for the period 2000–2003. Overall, the 90th percentile water Pb levels are lower in the early 2000s than in the early 1990s when the 1991 U.S. EPA Pb–Cu rule was implemented.

TABLE 6.49 Selected Drinking Water Pb Concentrations in U.S., English, and Dutch Plumbing Systems^{a,b,c,d}

Locale	Plumbing/Sample Conditions	Concentration ($\mu\text{g/l}$)		References
		Mean	Maximum	
Seattle, WA	Standing, overnight	4.3	11.5	Herrera et al. (1982)
University of Washington	Standing, overnight	3.9	170	Dangel (1975), as cited in Ohanian (1986)
Tolt River	Standing, overnight, 30 second flush	5	17	
Boston, MA	Composite sample	30	1,510	Karalekas et al. (1976)
Boston, MA	Standing, overnight	96	1,108	Worth et al. (1981)
	Running, 5-minute flush	17	208	
South Carolina	Metal pipes	20.7	163	Sandhu et al. (1977)
	Plastic pipes	18.7	73	
Morris County, NJ	Composite	44	260	Benson and Klein (1983)
England, various water lines for estate plumbing	Replacement Pb piping			Thomas et al. (1979)
	a. Standing, overnight	1,075	2,826	
	b. Running, 5-minute flush	139	354	
	Copper piping			
	a. Standing, overnight	4		
	b. Running, 5-minute flush	3		
The Netherlands	Composite samples	81	180	Zoetman and Haring (1978), as cited in Ohanian (1986)

^aAdapted from Ohanian (1986), covering various reports.^bVarious water Pb leaching conditions.^cU.S. tap water Pb action level (1991) = 15 $\mu\text{g/l}$ at 90th percentile community prevalence.^dWHO (1993) water Pb guideline = 10 $\mu\text{g/l}$.

Drinking Water Pb Concentrations in U.S., English,
and other countries^{a,b,c,d}

Sampling/Sample Conditions	Concentration ($\mu\text{g/l}$)		References
	Mean	Maximum	
Standing, overnight	4.3	11.5	Herrera et al. (1982)
Standing, overnight	3.9	170	Dangel (1975), as cited in Ohanian (1986)
Standing, overnight, and flush	5	17	
Tap water sample	30	1,510	Karalekas et al. (1976)
Standing, overnight	96	1,108	Worth et al. (1981)
Standing, 5-minute	17	208	
Lead pipes	20.7	163	Sandhu et al. (1977)
Galvanized pipes	18.7	73	
Tap water sample	44	260	Benson and Klein (1983)
Element Pb			
Standing, overnight	1,075	2,826	Thomas et al. (1979)
Standing, 5-minute flush	139	354	
Lead piping			
Standing, overnight	4		
Standing, 5-minute flush	3		Zoetman and Haring (1978), as cited in Ohanian (1986)
Tap water sample	81	180	
Tap water sample			

^acovering various reports.
^bconditions.

^c(1991) = 15 $\mu\text{g/l}$ at 90th percentile community prevalence.

^dmean = 10 $\mu\text{g/l}$.

TABLE 6.50 Illustrative Drinking Water Pb Levels Reported for U.S. and Canadian Communities

Locale	Occupancy Type	Water Status/Other	Pb-W ($\mu\text{g/l}$)	References
Midwestern United States	Residences	Standing Flushed	3.9 0.8	Clayton et al. (1999)
Massachusetts				
Gardner			25.0	Isaac et al. (1997)
Fall River			15.3	
New Bedford			11.6	
Clinton			7.7	
Arizona	Residences	—	0.7	Sofuoglu et al. (2003)
Halifax, Canada	Homes	Standing	16	Moir et al. (1996)
		Running	8	
	Apartments	Standing	3	
		Running	2	
	School	Standing	6	
		Running	5	
	Apartments		20	
	Houses		13	
Vancouver, Canada		Copper or plastic pipe		Singh and Mavinic (1991)

TABLE 6.51 Illustrative U.S. City Drinking Water Pb Levels at the 90th Percentile over Three Monitoring Periods^{a,b,c}

Locale	1992 90th %ile ($\mu\text{g/l}$)	1993 90th %ile ($\mu\text{g/l}$)	1999–2006 Monitoring Interval ($\mu\text{g/l}$)
Chicago, IL	20	10	7
Philadelphia, PA	15	322	13
Washington, DC	39	18	63
Detroit, MI	15	21	12
Minneapolis, MN	32	19	6
St. Paul, MN	28	54	11
Portland, OR	53	41	8
Phoenix, AZ	11	19	1
Yonkers, NY	110	68	18
Syracuse, NY	40	50	25
Galveston, TX	6	18	2
Miami Beach, FL	4	27	8
Richmond, VA	25	16	4
Tacoma, WA	17	32	12

^aAdapted from U.S. EPA (2006).^bCities are those exceeding the EPA 1991 action level in 1992 or 1993.^cRecent monitoring mainly in years 2000, 2001, 2002, 2003, except for one city in 1999–2001, one city in 2003–2006.

Percentages of water Pb collected through the day and lying within successively higher water Pb ranges have been reported in Great Britain. The data are in Table 6.52. The distribution statistics totaled for all three countries—England, Scotland, Wales—showed 25.3% of households in the 10–50 $\mu\text{g/l}$ range. The highest values, 301 $\mu\text{g/l}$ and above, were found for about 1% of samples in the three countries. The data for Scotland, with its historically very corrosive water supply and lead-containing water collection systems, were the main contributor to the high exceedences of Pb in drinking water. For example, 16% of Scottish water samples were in the range of 101–300 $\mu\text{g/l}$, and 5% were 301 $\mu\text{g/l}$ and above.

City Drinking Water Pb Levels at the 90th
Percentile Periods^{a,b,c}

90th Percentile	1993 90th Percentile ($\mu\text{g/l}$)	1999–2006 Monitoring Interval ($\mu\text{g/l}$)
	10	7
	322	13
	18	63
	21	12
	19	6
	54	11
	41	8
	19	1
	68	18
	50	25
	18	2
	27	8
	16	4
	32	12

^a1991 action level in 1992 or 1993.
^b2000, 2001, 2002, 2003, except for one city in 1999–2001.

collected through the day and lying within such ranges have been reported in Great Britain. The distribution statistics totaled for all three countries—showed 25.3% of households in the highest values, 301 $\mu\text{g/l}$ and above, were found for all three countries. The data for Scotland, with its tap water supply and lead-containing water collection system, contributor to the high exceedences of Pb in drinking water of Scottish water samples were in the range of 301 $\mu\text{g/l}$ and above.

TABLE 6.52 Late Twentieth Century Distribution of Lead Concentrations
in Tap Water Samples in Great Britain^a

Pb Level ($\mu\text{g/l}$)	Percent Households			
	England	Scotland	Wales	All
0–9	66.0	46.4	70.5	64.4
10–50	26.2	19.2	20.7	25.3
51–100	5.2	13.4	6.5	6.0
101–300	2.2	16.0	1.5	3.4
≥ 301	0.4	5.0	0.8	0.9
% Total	100.0	100.0	100.0	100.0

Source: WRC Water Research Centre (1983, Table 1).
^aDaytime water draws randomly consumed.

REFERENCES

- Adams, M.A., 1991. FDA total diet study: dietary intakes of lead and other chemicals. *Chem. Speciation Bioavailability* 3, 37–41.
- Adgate, J.L., Willis, R.D., Buckley, T.J., Chow, J.C., Watson, J.G., Rhoads, G.G., et al., 1998. Chemical mass balance source apportionment of lead in house dust. *Environ. Sci. Technol.* 32, 108–114.
- Al-Chalabi, A.S., Hawker, D., 1997. Response of vehicular lead to the presence of street dust in the atmospheric environment of major roads. *Sci. Total Environ.* 206, 195–202.
- Angle, C.R., McIntire, M.S., 1979. Environmental lead and children: the Omaha study. *J. Toxicol. Environ. Health* 5, 855–870.
- Angle, C.R., McIntire, M.S., 1982. Children, the barometer of environmental lead. *Adv. Pediatr.* 27, 3–31.
- Badawy, S.H., Helal, M.I.D., Chaudri, A.M., Lawlor, K., McGrath, S.P., 2002. Soil solid-phase controls lead activity in soil solution. *J. Environ. Qual.* 31, 162–167.
- Bartrop, D., Strehlow, C.D., Thornton, I., Webb, J.S., 1975. Absorption of lead from dust and soil. *Postgrad. Med. J.* 51, 801–804.
- Bates, M., Malcolm, M., Wyatt, R., Garrett, N., Galloway, Y., Speir, T., et al., 1995. Lead in children from older housing areas in the Wellington region. *N. Z. Med. J.* 108, 400–404.
- Beloian, A., 1985. Model system for use of dietary survey data to determine lead exposure from food. In: Mahaffey, K.R. (Ed.), *Dietary and Environmental Lead: Human Health Effects*. Elsevier, Amsterdam, the Netherlands, pp. 109–155.
- Benson, J.A., Klein, H., 1983. Lead in drinking water. Investigation of a corrosive water supply. *J. Environ. Health* 45, 179–181.
- Birden Jr., H.H., Calabrese, E.J., Stoddard, A., 1985. Lead dissolution from soldered joints. *J. Am. Water Works Assn.* 77, 66–70.

- Bolger, P.M., Carrington, C.D., Capar, S.G., Adams, M.A., 1991. Reductions in dietary lead exposure in the United States. *Chem. Speciation Bioavailability* 3, 31–36.
- Bornschein, R., Clark, S., Pan, W., Succop, P., 1991. Midvale community lead study. *Chem. Speciation Bioavailability* 3, 149–162.
- Brewer, R., Belzer, W., 2001. Assessment of metal concentrations in atmospheric particles from Barnaby Lake, British Columbia, Canada. *Atmos. Environ.* 35, 5223–5233.
- Brunekreef, B., Noy, D., Biersteker, K., Boleij, J., 1983. Blood lead levels of Dutch city children and their relationship to lead in the environment. *J. Air Pollut. Control Assoc.* 33, 872–876.
- Capar, S., 1990. Survey of lead and cadmium in adult canned foods eaten by young children. *J. Assoc. Off. Anal. Chem.* 73, 357–364.
- Capar, S., 1991. Analytical method aspects of assessing dietary intake of trace elements. In: Subramanian, K., Iyengar, G., Okamoto, K. (Eds.), *Biological Trace Element Research*. ACS Symp. Ser. 445, 181–195.
- Casteel, S., Weis, C.P., Henningsen, G.M., Brattin, W.J., 2006. Estimation of relative bioavailability of lead in soil and soil-like materials using young swine. *Environ. Health Perspect.* 114, 1162–1171.
- Chan, L.Y., Kwok, W.S., Chan, C.Y., 2000. Human exposure to respirable suspended particulate and airborne lead in different roadside microenvironments. *Chemosphere* 41, 93–99.
- Chaney, R., Mielke, H., 1986. Standards for soil lead limitations in the United States. *Trace Subst. Environ. Health* 20, 357–377.
- Chartsias, B., Colombo, A., Hatzichritidis, D., Leyendecker, W., 1986. The impact of gasoline lead and blood lead in man. *Sci. Total Environ.* 55, 275–282.
- Chen, J., Gao, J., 1993. The Chinese Total Diet Study in 1990. Part I. Chemical contaminants. *J. AOAC Int.* 76, 1193–1201.
- Chiaradia, M., Cuppelin, F., 2000. Behavior of airborne lead and temporal variation of its source effects in Geneva (Switzerland): comparison of anthropogenic versus natural processes. *Atmos. Environ.* 34, 959–971.
- Chirinje, T., Ma, L.Q., Reeves, M., Szulczewski, M., 2004. Lead distribution in near-surface soils of two Florida cities: Gainesville and Miami. *Geoderma* 119, 113–120.
- Chow, J.C., Watson, J.G., Ashbaugh, L.L., Magliano, K.L., 2003. Similarities and differences in PM₁₀ chemical source profiles for geological dust from the San Joaquin Valley, California. *Atmos. Environ.* 1317–1340.
- Clark, S., Bornschein, R.L., Succop, P., Que Hee, S.S., Hammond, P.B., Peace, B., 1985. Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. *Environ. Res.* 38, 46–53.
- Clark, S., Grote, J., Wilson, J., Succop, P., Chen, M., Galke, W., et al., 2004. Occurrence and determinants of increases in blood lead levels in children shortly after lead hazard control activities. *Environ. Res.* 96, 196–205.
- Clayton, C.A., Pellizzari, E.D., Whitmore, R.W., Perritt, R.L., Quackenboss, J.J., 1999. National human exposure assessment survey (NHEXAS): distributions and associations of lead, arsenic, and volatile organic compounds in EPA Region 5. *J. Exposure Anal. Environ. Epidemiol.* 9, 381–392.
- Colorado Department of Health, 1990. Leadville Metals Exposure Study. Division of Disease Control and Environmental Epidemiology. With University of Colorado at Denver and the U.S. Agency for Toxic Substances and Disease Registry, April.
- Culbard, E.B., Thornton, L., Watt, J., Wheatley, M., Moorcroft, S., Thompson, M., 1988. Metal contamination in British suburban dusts and soils. *J. Environ. Qual.* 17, 226–234.

- Capar, S.G., Adams, M.A., 1991. Reductions in dietary lead. *Chem. Speciation Bioavailability* 3, 31–36.
- W., Succop, P., 1991. Midvale community lead study. *Chem.* 149–162.
- assessment of metal concentrations in atmospheric particles from
bia, Canada. *Atmos. Environ.* 35, 5223–5233.
- eker, K., Boleij, J., 1983. Blood lead levels of Dutch city
p to lead in the environment. *J. Air Pollut. Control Assoc.* 33.
- and cadmium in adult canned foods eaten by young children.
3, 357–364.
- od aspects of assessing dietary intake of trace elements. In:
G., Okamoto, K. (Eds.), *Biological Trace Element Research*.
95.
- en, G.M., Brattin, W.J., 2006. Estimation of relative bioavail-
il-like materials using young swine. *Environ. Health Perspect.*
- C.Y., 2000. Human exposure to respirable suspended particulate
roadside microenvironments. *Chemosphere* 41, 93–99.
- standards for soil lead limitations in the United States. *Trace*
7–377.
- ichritidis, D., Leyendecker, W., 1986. The impact of gasoline
Sci. Total Environ. 55, 275–282.
- ese Total Diet Study in 1990. Part I. Chemical contaminants.
- Behavior of airborne lead and temporal variation of its source
and): comparison of anthropogenic versus natural processes.
- M., Szulcowski, M., 2004. Lead distribution in near-surface
inesville and Miami. *Geoderma* 119, 113–120.
- gh, L.L., Magliano, K.L., 2003. Similarities and differences in
s for geological dust from the San Joaquin Valley, California.
- ccop, P., Que Hee, S.S., Hammond, P.B., Peace, B., 1985.
g as an indicator of potential environmental lead exposure and
Environ. Res. 38, 46–53.
- uccop, P., Chen, M., Galke, W., et al., 2004. Occurrence and
blood lead levels in children shortly after lead hazard control
96–205.
- hitmore, R.W., Perritt, R.L., Quackenboss, J.J., 1999. National
urvey (NHEXAS): distributions and associations of lead, arse-
compounds in EPA Region 5. *J. Exposure Anal. Environ.*
1990. Leadville Metals Exposure Study. Division of Disease
pidemiology. With University of Colorado at Denver and the
nces and Disease Registry, April.
- J., Wheatley, M., Moorcroft, S., Thompson, M., 1988. Metal
urban dusts and soils. *J. Environ. Qual.* 17, 226–234.
- Dabeka, R.W., McKensie, A.D., Lacroix, G.M.A., 1987. Dietary intakes of lead, cadmium, arse-
nic and fluoride by Canadian adults: a 24-hour duplicate diet study. *Food Addit. Contam.* 4,
89–102.
- Davidson, C.I., Chu, L., Grimm, T.C., Nasta, M.A., Qamoos, M.P., 1981. Wet and dry deposi-
tion of trace elements onto the Greenland ice sheet. *Atmos. Environ.* 15, 1429–1437.
- Davies, B.E., 1983. A graphical estimation of the normal lead content of some British soils.
Geoderma 29, 67–75.
- Davies, D.J.A., Watt, J.M., Thornton, I., 1987. Air lead concentrations in Birmingham,
England—a comparison between levels inside and outside inner-city homes. *Environ.*
Geochem. Health 9, 3–7.
- Day, J.P., Hart, M., Robinson, M.S., 1975. Lead in urban street dust. *Nature* 253, 343–345.
- Del Delumyea, R., Kalivretenos, A., 1987. Elemental carbon and lead content of fine particles
from American and French cities of comparable size and industry, 1985. *Atmos. Environ.*
21, 1643–1647.
- DeMiguel, E., Llamas, J.F., Chacon, E., Berg, T., Larssen, S., Royset, O., et al., 1997. Origin
and patterns of distribution of trace elements in street dust: unleaded petrol and urban lead.
Atmos. Environ. 31, 2733–2740.
- DeMora, S.J., Harrison, R., 1984. Lead in tap water: contamination and chemistry. *Chem. Br.*
900–906.
- Deroanne-Bauvin, J., Delcarte, E., Impins, R., 1987. Monitoring of lead composition near high-
ways—a ten year study. *Sci. Total Environ.* 59, 257–266.
- Diemel, J.A.L., Brunekreef, B., Boleij, J.S.M., Biersteker, K., Veenstra, S.J., 1981. The Arnhem
lead study: II. Indoor pollution and indoor/outdoor relationships. *Environ. Res.* 25, 449–456.
- Ducoffre, G., Claeys, F., Bruaux, P., 1990. Lowering time trend of blood lead levels in Belgium
since 1978. *Environ. Res.* 51, 25–34.
- Dudka, S., Miller, W.P., 1999. Accumulation of potentially toxic elements in plants and their
transfer to human food chains. *J. Environ. Sci. Health, Part B* 34, 681–708.
- Durum, W.H., Hem, J.D., Heidl, S.G., 1971. Reconnaissance of selected minor elements in sur-
face waters of the United States. USGS Circular No. 643. United States Geological Survey,
Washington, DC.
- Dzubay, T.G., Hines, L.E., Stevens, R.K., 1976. Particle bounce errors in cascade impactors.
Atmos. Environ. 10, 229–234.
- Elinder, C.-G., Friberg, L., Lind, B., 1986. Decreased blood levels in residents of Stockholm for
the period 1970–1984. *Scand. J. Work Environ. Health.* 12, 114–120.
- El-Shobokshy, M.S., 1985. The dependence of airborne particulate deposition on atmospheric
stability and surface conditions. *Atmos. Environ.* 19, 1191–1197.
- Erel, Y., Veron, A., Halicz, L., 1997. Tracing the transport of anthropogenic lead in the atmo-
sphere and in soils using isotopic ratios. *Geochim. Cosmochim. Acta* 58, 4495–4505.
- Erel, Y., Axelrod, T., Veron, A., Mahrer, Y., Katsafados, P., Dayan, U., 2002. Transboundary
atmospheric lead pollution. *Environ. Sci. Technol.* 36, 3230–3233.
- Ewers, L., Clark, S., Menrath, W., Succop, P., Bornschein, R., 1994. Clean-up of lead in house-
hold carpet and floor dust. *Am. Ind. Hyg. Assoc. J.* 55, 650–657.
- Facchetti, S., 1989. Lead in petrol. The isotopic lead experiment. *Acc. Chem. Res.* 22, 370–374.
- Facchetti S., Geiss F., 1982. Isotopic lead experiment: status report. Commission of the
European Communities, Publication No. Eur 8352 EN. CEC, Luxembourg.
- Farfel, M.R., Bannon, D., Chisolm Jr., J.J., Lees, P.S.J., Lim, B.S., Rohde, C.A., 1994.
Comparison of a wipe and a vacuum collection method for the determination of lead in resi-
dential dusts. *Environ. Res.* 65, 291–301.

- Fergusson, J.E., Ryan, D.E., 1984. The elemental composition of street dust from large and small urban areas related to city type, source and particle size. *Sci. Total Environ.* 34, 101–116.
- Fergusson, J.E., Schroeder, R.J., 1985. Lead in house dust of Christchurch, New Zealand. *Sci. Total Environ.* 46, 61–72.
- Finster, M.E., Gray, K., Binns, H.J., 2004. Lead levels of edibles grown in contaminated residential soils: a field survey. *Sci. Total Environ.* 320, 245–257.
- Freitas, H., Prasad, M.N.V., Pratas, J., 2004. Plant community tolerant to trace elements growing on the degraded soils of Sao Domingos mine in the south east of Portugal: environmental implications. *Environ. Int.* 30, 65–72.
- Fugas, M., Wilder, B., Paukovic, R., Hrsak, J., Steiner-Skreb, D., 1973. Concentration levels and particle size distribution of lead in the air of an urban and an industrial area as a basis for the calculation of population exposure. In: Barth, D., Berlin, A., Engel, R., Reicht, P., Smeets, J. (Eds.), *The Environmental Health Aspects of Lead: Proceedings of an International Symposium, October, 1972, Amsterdam, the Netherlands. Commission of the European Communities, Luxembourg*, pp. 961–968.
- Galal-Gorchev, H., 1991. Global overview of dietary lead exposure. *Chem. Speciation Bioavailability* 3, 5–11.
- Galke, W.A., Hammer, D.I., Keil, J.E., Lawrence, S.W., 1975. Environmental determinants of lead burdens in children. In: Hutchinson, T.C., Epstein, S., Page, A.L., Van Loon, J. (Eds.), *International Conference on Heavy Metals in the Environment. Proceedings*, vol. 3. Toronto, Canada. Institute for Environmental Studies, pp. 53–74.
- GEMS-Food, 1991. Summary of 1986–1988 monitoring data. FAO/WHO Collaborating Centres for Food Contamination Monitoring. Geneva, Switzerland.
- Gillies, J.A., O'Connor, C.M., Mamane, Y., Gertler, A.W., 1999. Chemical profiles for characterizing dust sources in an urban area, western Nevada, USA. In: Livingstone, I., (Ed.), *Aeolian geomorphology. Papers from the 4th International Conference on Aeolian Research*, 1998. Oxford, United Kingdom. *Z. Geomorph.* 116 (Suppl.), 19–44.
- Godin, P.M., Feinberg, M.H., Ducauze, C.J., 1985. Modeling of soil contamination by airborne lead and cadmium around several emission sources. *Environ. Pollut. Ser. B* 10, 97–114.
- Gulson, B.L., Mahaffey, K.R., Mizon, K.J., Korsch, M.J., Cameron, M.A., Vimpani, G., 1995. Contribution of tissue lead to blood lead in adult female subjects based on stable lead isotope methods. *J. Lab. Clin. Med.* 125, 703–712.
- Gulson, B.L., Jameson, C.W., Mahaffey, K.R., Mizon, K.J., Korsch, M.J., Vimpani, G., 1997. Pregnancy increases mobilization of lead from maternal skeleton. *J. Lab. Clin. Med.* 130, 51–62.
- Harrison, R.M., 1979. Toxic metals in street and household dusts. *Sci. Total Environ.* 11, 89–97.
- Harrison, R.M., Tilling, R., Callen Romero, M.S., Harrad, S., Jarvis, K., 2003. A study of trace metals and polycyclic aromatic hydrocarbons in the roadside environment. *Atmos. Environ.* 37, 2391–2402.
- Hartwell, T.D., Handy, R.W., Harris, B.S., Williams, S.R., Gehlbach, S.H., 1983. Heavy metal exposure in populations living around zinc and copper smelters. *Arch. Environ. Health* 38, 284–295.
- Herrera, C.E., Ferguson, J.F., Benjamin, M.M., 1982. Evaluating the potential for contaminating drinking water from the corrosion of tin-antimony solder. *J. Am. Water Works Assn.* 74, 368–375.
- Hertzman, C., Ward, H., Ames, N., Kelly, S., Yates, C., 1991. Childhood lead exposure in Trail revisited. *Can. J. Public Health* 82, 385–391.

4. The elemental composition of street dust from large and small type, source and particle size. *Sci. Total Environ.* 34, 101–116.
- , 1985. Lead in house dust of Christchurch, New Zealand. *Sci. Total Environ.* 320, 245–257.
- H.J., 2004. Lead levels of edibles grown in contaminated residential areas. *Sci. Total Environ.* 320, 245–257.
- tas, J., 2004. Plant community tolerant to trace elements growing at Domingos mine in the south east of Portugal: environmental assessment. *Environ. Pollut.* 100, 65–72.
- , R., Hrsak, J., Steiner-Skreb, D., 1973. Concentration levels and lead in the air of an urban and an industrial area as a basis for exposure. In: Barth, D., Berlin, A., Engel, R., Rechi, P., *Environmental Health Aspects of Lead: Proceedings of an International Conference, October, 1972, Amsterdam, the Netherlands, Commission of the European Communities*, pp. 961–968.
- Global overview of dietary lead exposure. *Chem. Speciation Reviews*, pp. 961–968.
- eil, J.E., Lawrence, S.W., 1975. Environmental determinants of lead exposure. In: Hutchinson, T.C., Epstein, S., Page, A.L., Van Loon, J. (Eds.), *Heavy Metals in the Environment. Proceedings, vol. 3. Toronto, Environmental Studies*, pp. 53–74.
- 1986–1988 monitoring data. FAO/WHO Collaborating Centres for the Monitoring of Lead in the Environment. Geneva, Switzerland.
- Mamane, Y., Gertler, A.W., 1999. Chemical profiles for characterization of lead in an urban area, western Nevada, USA. In: Livingstone, I. (Ed.), *Lead in the Environment: Proceedings from the 4th International Conference on Aeolian Research, 1999, Z. Geomorph.* 116 (Suppl.), 19–44.
- ucauze, C.J., 1985. Modeling of soil contamination by airborne lead from several emission sources. *Environ. Pollut. Ser. B* 10, 97–114.
- Mizon, K.J., Korsch, M.J., Cameron, M.A., Vimpani, G., 1995. Blood lead in adult female subjects based on stable lead isotope ratios. *Med.* 125, 703–712.
- Mahaffey, K.R., Mizon, K.J., Korsch, M.J., Vimpani, G., 1997. Determination of lead from maternal skeleton. *J. Lab. Clin. Med.* 130, 51–62.
- metals in street and household dusts. *Sci. Total Environ.* 11, 101–116.
- en Romero, M.S., Harrad, S., Jarvis, K., 2003. A study of trace organic hydrocarbons in the roadside environment. *Atmos. Environ.* 37, 1023–1032.
- arris, B.S., Williams, S.R., Gehlbach, S.H., 1983. Heavy metal contamination around zinc and copper smelters. *Arch. Environ. Health* 38, 1023–1032.
- enjamin, M.M., 1982. Evaluating the potential for contaminating the environment of tin-antimony solder. *J. Am. Water Works Assn.* 74, 1023–1032.
- N., Kelly, S., Yates, C., 1991. Childhood lead exposure in Trail, British Columbia. *Health Perspect.* 101, 598–616.
- Hills, S.R., 2003. Effect of smelter emission reductions on children's blood lead levels. *Sci. Total Environ.* 303, 51–58.
- Ho, K.F., Lee, S.C., Chow, J.C., Watson, J.G., 2003. Characterization of PM₁₀ and PM_{2.5} source profiles for fugitive dust in Hong Kong. *Atmos. Environ.* 37, 1023–1032.
- Hui, C.A., 2002. Concentrations of chromium, manganese, and lead in air and in avian eggs. *Environ. Pollut.* 120, 201–206.
- Hunt, W.F., Pinkerton, C., McNulty, O., Creason, J., 1971. A study in trace element pollution of air in 77 midwestern cities. *Trace Subst. Environ. Health* 4, 56–58.
- Isaac, R.A., Gil, L., Cooperman, A.N., Hulme, K.I., Eddy, B., Ruiz, M., et al., 1997. Corrosion in drinking water distribution systems: a major contributor to copper and lead in wastewater and effluents. *Environ. Sci. Technol.* 31, 3198–3203.
- Jacobs, D.E., Clickner, R., Zhou, J., Viet, S.M., Marker, D.A., Rogers, J.W., et al., 2002. The prevalence of lead-based paint hazards in U.S. housing. *Environ. Health Perspect.* 110, A599–A606.
- Jensen, R.A., Laxen, D.P.H., 1985. Sources of lead in urban dust: identification of a contribution from newspaper printworks. *Sci. Total Environ.* 46, 19–27.
- Joberg, D.R., Kleinman, C.F., Kwon, S.C., 1997. Position paper of the American Council on Science and Health: lead and human health. *Ecotoxicol. Environ. Saf.* 38, 162–180.
- Kapaki, E.N., Varelas, P.N., Syrigou, A.I., Spanaki, M.V., Andreadou, E., Kakami, A.E., et al., 1998. Blood lead levels of traffic- and gasoline exposed professionals in the city of Athens. *Arch. Environ. Health* 53, 286–291.
- Karalekas, P.C., Craun, G.F., Hammonds, A.F., 1976. Lead and other trace metals in drinking water in the Boston metropolitan area. *J. Am. Water Works Assn.* 90, 150–172.
- Kimbrough, D.E., Suffet, I.H., 1995. Off-site forensic determination of airborne elemental emissions by multi-media analysis: a case study of two secondary lead smelters. *Environ. Sci. Technol.* 29, 2217–2221.
- Kurkjian, R., Dunlap, C., Flegal, A.R., 2002. Lead isotope tracking of atmospheric response to post-industrial conditions in Yerevan, Armenia. *Atmos. Environ.* 36, 1421–1429.
- Landrigan, P.J., Gehlbach, S.H., Rosenblum, B.F., Shoults, J.M., Candelaria, R.M., Barthel, W.F., et al., 1975. Epidemic lead absorption near an ore smelter: the role of particulate lead. *N. Engl. J. Med.* 292, 123–129.
- Landrigan, P.J., Baker Jr., E.L., Himmelstein, J.S., Stein, G.F., Wedding, J.P., Straub, W.E., 1982. Exposure to lead from the Mystic River bridge: the dilemma of deleading 306, 673–676. *N. Engl. J. Med.* 306, 673–676.
- Lanphear, B.P., Edmond, M., Jacobs, D.E., Weitzman, M., Tanner, M., Winter, N.L., et al., 1995. A side-by-side comparison of dust collection methods for sampling lead-contaminated house dust. *Environ. Res.* 68, 114–123.
- Lanphear, B.P., Weitzman, M., Winter, N.L., Eberly, S., Yakir, B., Tanner, M., et al., 1996. Lead-contaminated house dust and urban children's blood lead levels. *Am. J. Public Health* 86, 1416–1421.
- Lau, W.M., Wong, H.M., 1982. An ecological survey of lead contents in roadside dusts and soils in Hong Kong. *Environ. Res.* 28, 39–54.
- Laxen, D.P.H., Raab, G.M., Fulton, M., 1987. Children's blood lead and exposure to lead in household dust and water—a basis for an environmental standard for lead in dust. *Sci. Total Environ.* 66, 235–244.
- Leggett, R.W., 1993. An age-specific kinetic model of lead metabolism in humans. *Environ. Health Perspect.* 101, 598–616.

- Linzon, S.N., Chai, B.L., Temple, P.J., Pearson, R.G., Smith, M.L., 1976. Lead contamination in urban soils and vegetation by emissions from secondary lead industries. *J. Air. Pollut. Control Assoc.* 26, 650-654.
- Liu, Z.P., 2003. Lead poisoning combined with cadmium in sheep and horses in the vicinity of non-ferrous metal smelters. *Sci. Total Environ.* 309, 117-126.
- Loo, B.W., Adachi, R.S., Cork, C.P., Goulding, S.S., Jaklevic, J.N., Landis, D.A., et al., 1979. A second generation dichotomous sampler for large-scale monitoring of airborne particulate matter. Lawrence Berkeley Laboratory. Report No. 8725. As cited in U.S. EPA 1986a, Ch. 4.
- Maenhaut, W., Zoller, W.H., Duce, R.A., Hoffman, G.L., 1979. Concentration and size distribution of particulate trace elements in the south polar atmosphere. *J. Geophys. Res.* 84, 2421-2431.
- Malcoe, L.H., Lynch, R.A., Kegler, M.C., Skaggs, V.J., 2002. Lead sources, behaviors, and socioeconomic factors in relation to blood lead of Native American and white children: a community-based assessment of a former mining area. *Environ. Health Perspect.* 110 (Suppl. 2), 221-231.
- Markowitz, M.E., Weinberger, H.L., 1990. Immobilization-related lead toxicity in previously lead-poisoned children. *Pediatrics* 86, 455-457.
- McGrath, S.P., 1986. The range of metal concentrations in topsoils of England and Wales in relation to soil protection guidelines. In: Hemphill, D.D. (Ed.), *Trace Substances in Environmental Health*, 20, pp. 242-252.
- McIntire, M.S., Angle, C.R., 1973. Air lead/blood lead in G-6-PD deficient black school children. In: Barth, D., Berlin, A., Engel, R., Recht, P., Smeets, J. (Eds.), *Environmental Health Aspects of Lead. Proceedings of an International Symposium, October, 1972, Amsterdam, the Netherlands. Commission of the European Communities, Luxembourg*, pp. 749-761.
- McKeague, J.A., Wolynetz, M.S., 1980. Background levels of minor elements in some Canadian soils. *Geoderma* 24, 299-307.
- Milar, C.R., Mushak, P., 1982. Lead-contaminated house dust: hazard, management, and decontamination. In: Chisolm, J.J. Jr., O'Hara, D.M. (Eds.), *Lead Absorption in Children: Management, Clinical, and Environmental Aspects. Proceedings of a Conference, November, 1979. Urban and Schwarzenberg, Baltimore, MD*, pp. 143-152.
- Mitchell, D.G., Aldous, K.M., 1974. Lead content of foodstuffs. *Environ. Health Perspect.* 7, 59-64.
- Moir, C.M., Freedman, B., McCurdy, R., 1996. Metal mobilization from water-distribution systems of buildings serviced by lead-pipe mains. *Can. Water Resour. J.* 21, 45-52.
- Moseholm, L., Larsen, E.H., Andersen, B., Nielsen, M.M., 1992. Atmospheric deposition of trace elements around point sources and human health risk assessment. I. Impact zones near a source of lead emissions. *Sci. Total Environ.* 126, 243-262.
- Murgueyio, A.M., Evans, R.G., Daryl, R., 1998. Relationship between soil and dust lead in a lead mining area and blood lead levels. *J. Exposure Anal. Environ. Epidemiol.* 8, 173-186.
- Mushak, P., 1991. Gastrointestinal absorption of lead in children and in adults: overview of biological and biophysico-chemical aspects. *Chem. Speciation Bioavailability* 3, 87-104.
- Mushak, P., 1993. New directions in the toxicokinetics of human lead exposure. *Neurotoxicology* 14, 29-42.
- Mushak, P., 1998. Uses and limits of empirical data in measuring and modeling human lead exposure. *Environ. Health Perspect.* 106 (Suppl. 6), 1467-1484.

- P.J., Pearson, R.G., Smith, M.L., 1976. Lead contamination in emissions from secondary lead industries. *J. Air Pollut. Combined with cadmium in sheep and horses in the vicinity of*. *Total Environ.* 309, 117-126.
- E.P., Goulding, S.S., Jaklevic, J.N., Landis, D.A., et al., 1979. Continuous sampler for large-scale monitoring of airborne particulate Laboratory. Report No. 8725. As cited in U.S. EPA
- e, R.A., Hoffman, G.L., 1979. Concentration and size distributions in the south polar atmosphere. *J. Geophys. Res.* 84,
- er, M.C., Skaggs, V.J., 2002. Lead sources, behaviors, and tion to blood lead of Native American and white children: a of a former mining area. *Environ. Health Perspect.* 110
- L., 1990. Immobilization-related lead toxicity in previously ics 86, 455-457.
- of metal concentrations in topsoils of England and Wales in uidelines. In: Hemphill, D.D. (Ed.), *Trace Substances in* 242-252.
73. Air lead/blood lead in G-6-PD deficient black school , A., Engel, R., Recht, P., Smeets, J. (Eds.), *Environmental* Proceedings of an International Symposium, October, 1972, Commission of the European Communities, Luxembourg.
980. Background levels of minor elements in some Canadian
- d-contaminated house dust: hazard, management, and decon- Jr., O'Hara, D.M. (Eds.), *Lead Absorption in Children: Environmental Aspects. Proceedings of a Conference,* Schwartzberg, Baltimore, MD, pp. 143-152.
4. Lead content of foodstuffs. *Environ. Health Perspect.* 7,
- ly, R., 1996. Metal mobilization from water-distribution sys- lead-pipe mains. *Can. Water Resour. J.* 21, 45-52.
- ersen, B., Nielsen, M.M., 1992. Atmospheric deposition of sources and human health risk assessment. I. Impact zones near *Total Environ.* 126, 243-262.
- ryl, R., 1998. Relationship between soil and dust lead in a l levels. *J. Exposure Anal. Environ. Epidemiol.* 8, 173-186.
- bsorption of lead in children and in adults: overview of bio- aspects. *Chem. Speciation Bioavailability* 3, 87-104.
- ions in the toxicokinetics of human lead exposure.
- of empirical data in measuring and modeling human lead ect. 106 (Suppl. 6), 1467-1484.
- Mushak, P., Crocetti, A.F., 1990. Methods for reducing lead exposure in young children and other risk groups: an integrated summary of a report to the U.S. Congress on childhood lead poisoning. *Environ. Health Perspect.* 89, 125-135.
- Mushak, P., Mushak, E.W., 2000. Lessons learned: a comparative analysis of the evolution of lead and mercury as public health hazards. In: Nriagu, J.O. (Ed.), 11th International Conference on Heavy Metals in the Environment. CD-ROM: Manuscript No. 1445. University of Michigan, Ann Arbor, MI: August 6-10, 2000.
- Mushak, P., Schroeder, C., 1980. Multiple media pollutant exposures and their regulation. Report to the National Commission on Air Quality. Contract No. 23a-AQ-6981. Discussed and cited in the 1981 National Commission on Air Quality report: To Breathe Clean Air, pp. 2.1.9, 2.2-4, 3.1-30, 3.1-34.
- National Academy of Sciences: National Research Council, 1980. Lead in the Human Environment. National Academy Press, Washington, DC.
- National Academy of Sciences: National Research Council, 1993. Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations. National Academy Press, Washington, DC.
- National Air Pollution Surveillance Reports (NAPS), 1971-1976. Report Series No. EPS-5-AP. Air Pollution Control Directorate, Ottawa, Canada.
- Needleman, H.L., Davidson, I., Sewell, E.M., Shapiro, I.M., 1974. Subclinical lead exposure in Philadelphia school children: identification by dentine lead analysis. *N. Engl. J. Med.* 290, 245-258.
- Neri, L.C., Johansen, H.L., Schmitt, N., Pagan, R.T., Hewitt, D., 1978. Blood lead levels in children in two British Columbia communities. *Trace Subst. Environ. Health* 12, 403-410.
- Nriagu, J.O., 1978. Lead in the atmosphere. In: Nriagu, J.O. (Ed.), *The Biogeochemistry of Lead in the Environment: Part A, Ecological Cycles.* Elsevier/North Holland Biomedical Press, Amsterdam, the Netherlands, pp. 137-184.
- Nriagu, J.O., 1979. Global inventory of natural and anthropogenic emissions of trace metals to the atmosphere. *Nature* 279, 409-411.
- Nriagu, J.O., 1983a. Lead and Lead Poisoning in Antiquity. John Wiley & Sons, New York.
- Nriagu, J.O., 1983b. Saturnine gout among Roman aristocrats: did lead poisoning contribute to the fall of the Empire? *N. Engl. J. Med.* 308, 660-663.
- Nriagu, J.O., 1985. Historical perspective on the contamination of food and beverages with lead. In: Mahaffey, K.R. (Ed.), *Dietary and Environmental Lead: Human Health Effects.* Elsevier, New York, pp. 1-41.
- Nutrition Foundation, 1982. Assessment of the safety of lead and lead salts in food: a Report of the Nutrition Foundation's Expert Advisory Committee, Washington, DC.
- O'Flaherty, E.J., 1998. A physiologically based kinetic model for lead in children and adults. *Environ. Health Perspect.* 106 (Suppl. 6), 1495-1503.
- Ohanian, E.V., 1986. Health effects of corrosion products in drinking water. *Trace Subst. Environ. Health* 20, 122-138.
- O'Heany, J., Kusiak, R., Duncan, C.E., Smith, L.F., Spielberg, L., 1988. Blood lead and associated risk factors in Ontario children. *Sci. Total Environ.* 71, 477-483.
- Oliver, D.P., McLaughlin, M.J., Naidu, R., Smith, L.H., Maynard, E.J., Calder, I.C., 1999. Measuring lead bioavailability from household dusts using an in vitro model. *Environ. Sci.* 33, 4434-4439.
- Page, R.A., Cawse, P.A., Baker, S.J., 1988. The effect of reducing petrol lead on airborne lead in Wales, U.K.. *Sci. Total Environ.* 68, 71-77.

- Pao, E.M., 1989. Obtaining data on food intake. In: Livingston, G.E. (Ed.), *Nutritional Status Assessment of the Individual*. Food and Nutrition Press, Trumbull, CT.
- Patterson, C.C., 1982. Natural Levels of Lead in Humans. Institute for Environmental Studies, University of North Carolina, Chapel Hill, NC, Carolina, Environmental Essay Series, vol.3.
- Patterson, C.C., 1983. Criticism of "flow of metals into the global atmosphere." *Geochim. Cosmochim. Acta* 47, 1163-1168.
- Pennington, J.A.T., 1983. Revision of the Total Diet Study food list and diet. *J. Am. Diet. Assoc.* 82, 166-173.
- Pennington, J.A.T., Gunderson, E., 1987. History of the Food and Drug Administration's Total Diet Study—1961 to 1987. *J. Assoc. Off. Anal. Chem.* 70, 772-782.
- Pinkerton, C., Creason, J.P., Hammer, D.I., Colucci, A.V., 1973. Multi-media indices of environmental trace-metal exposure in humans. In: Hoekstra, W.G., Suttie, J.W., Ganther, H.E., Mertz, W. (Eds.), *Trace Element Metabolism in Animals—2: Proceedings of the 2nd International Symposium on Trace Element Metabolism in Animals*, Madison, WI. University Park Press, Baltimore, MD, pp. 465-469.
- Ponka, A., Salminen, E., Ahonen, S., 1991. Lead in the ambient air and blood specimens of children in Helsinki. *Sci. Total Environ.* 138, 301-308.
- Rabinowitz, M.B., Bellinger, D.C., 1988. Soil lead-blood lead relationship among Boston children. *Bull. Environ. Contam. Toxicol.* 41, 791-797.
- Rabinowitz, M.B., Wetherill, G.W., Kopple, J.D., 1976. Kinetic analysis of lead metabolism in healthy humans. *J. Clin. Invest.* 58, 260-270.
- Rameau, J.T.L.B., 1973. Lead as an environmental pollutant. In: Barth, D., Berlin, A., Engel, R., Recht, P., Smeets, J. (Eds.), *Environmental Health Aspects of Lead. Proceedings of an International Symposium, October, 1972, Amsterdam, the Netherlands. Commission of the European Communities, Luxembourg*, pp. 189-200.
- Rieuwerts, J.S., Farago, M., 1996. Heavy metal pollution in the vicinity of a secondary lead smelter in Czech Republic. *Appl. Geochem.* 11, 17-23.
- Roels, H.A., Buchet, J.P., Lauwerys, R.R., Bruaux, P., Claeys-Thoreau, F., Lafontaine, A., et al., 1980. Exposure to lead by the oral and pulmonary routes of children living in the vicinity of a primary lead smelter. *Environ. Res.* 22, 81-94.
- Samuels, E.R., Méranger, J.C., 1984. Preliminary studies on the leaching of some metals from kitchen faucets. *Water Res.* 18, 75-80.
- Sandhu, S.S., Warren, W.J., Nelson, P., 1977. Inorganic contaminants in rural drinking water. *J. Am. Water Works Assoc.* 69, 219-222.
- Schroeder, H.A., Balassa, J.J., 1961. Abnormal trace metals in man: Lead. *J. Chronic Dis.* 14, 408-425.
- Settle, D.M., Patterson, C.C., 1980. Lead in albacore: guide to lead pollution in the Americas. *Science* 207, 1167-1176.
- Settle, D.M., Patterson, C.C., 1982. Magnitude and sources of precipitation and dry deposition fluxes of industrial and natural leads to the North Pacific at Eniwetok. *J. Geophys. Res.* 87, 8857-8869.
- Shacklette, H.T., Hamilton, J.C., Boerngen, J.G., Bowles, J.M., 1971. Elemental composition of surficial materials in the conterminous United States: an account of the amounts of certain elements in samples of soils and other regoliths. Geological Survey Professional Paper No. 574-D. U.S. Geological Survey, Washington, DC.
- Schilling, R.J., Bain, R.P., 1988. Prediction of children's blood lead levels on the basis of household-specific soil lead levels. *Am. J. Epidemiol.* 128, 197-205.

- food intake. In: Livingston, G.E. (Ed.), *Nutritional Status and Nutrition Press*, Trumbull, CT.
- of Lead in Humans. Institute for Environmental Studies, Chapel Hill, NC, Carolina, Environmental Essay Series, vol. 3, "flow of metals into the global atmosphere." *Geochim.* 8.
- of the Total Diet Study food list and diet. *J. Am. Diet.* 1987. History of the Food and Drug Administration's Total Diet Study. *Off. Anal. Chem.* 70, 772-782.
- Colucci, A.V., 1973. Multi-media indices of environmental contamination in humans. In: Hoekstra, W.G., Suttie, J.W., Ganther, H.E., *Trace Element Metabolism in Animals—2: Proceedings of the 2nd Trace Element Metabolism in Animals*, Madison, WI, pp. 465-469.
1991. Lead in the ambient air and blood specimens of children. *Environ.* 138, 301-308.
1988. Soil lead-blood lead relationship among Boston children. *Toxicol.* 41, 791-797.
- Kopple, J.D., 1976. Kinetic analysis of lead metabolism in children. *Environ. Health Perspect.* 58, 260-270.
- environmental pollutant. In: Barth, D., Berlin, A., Engel, R., *Environmental Health Aspects of Lead*. Proceedings of an international symposium, 1972, Amsterdam, the Netherlands. Commission of the European Communities, Luxembourg, pp. 189-200.
- Heavy metal pollution in the vicinity of a secondary lead smelter. *Environ. Geochem.* 11, 17-23.
1984. Preliminary studies on the leaching of some metals from lead-contaminated soils. *Environ. Res.* 22, 81-94.
1977. Inorganic contaminants in rural drinking water. *Environ. Res.* 19, 219-222.
1961. Abnormal trace metals in man: Lead. *J. Chronic Dis.* 14, 1-10.
1980. Lead in albacore: guide to lead pollution in the Americas. *Environ. Res.* 22, 81-94.
1982. Magnitude and sources of precipitation and dry deposition of lead to the North Pacific at Eniwetok. *J. Geophys. Res.* 87, 10,000-10,010.
- Boerger, J.G., Bowles, J.M., 1971. Elemental composition of rocks from the conterminous United States: an account of the amounts of certain elements and other regoliths. Geological Survey Professional Paper No. 1000, Washington, DC.
- Prediction of children's blood lead levels on the basis of household lead levels. *Am. J. Epidemiol.* 128, 197-205.
- Silvergeld, E.K., Schwartz, J., Mahaffey, K.R., 1988. Lead and osteoporosis: mobilization of lead from bone to blood in postmenopausal women. *Environ. Res.* 47, 79-94.
- Singh, I., Mavrin, D.S., 1991. Significance of buildings and plumbing specifics on trace metal concentrations in drinking water. *Can. J. Civ. Eng.* 18, 893-903.
- Sofuoglu, S.C., Lebowitz, M.D., O'Rourke, M.K., Robertson, G.L., Dellarco, M., Moschandreas, D.J., 2003. Exposure and risk estimates for Arizona drinking water. *J. Am. Water Works Assoc.* 95, 67-79.
- Stark, A.D., Quah, R.F., Meigs, J.W., DeLouise, E.R., 1982. The relationship of environmental lead to blood-lead levels in children. *Environ. Res.* 27, 372-383.
- Stambeck, J., Sjödin, A., Andreasson, K., 2002. Metal emissions from road traffic and the influence of resuspension—results from two tunnel studies. *Atmos. Environ.* 36, 4735-4744.
- Stewart, D.D., 1887. Notes on some obscure cases of poisoning by lead chromate; manifested chiefly by encephalopathy. *Medical News* 18 (June), 676-681.
- Stewart, D.D., 1895. Lead convulsions. A study of sixteen cases. *Am. J. Med. Sci.* CLX, 286-306.
- Subramanian, K.S., Sastri, V.S., Connor, J.W., 1994. Drinking water quality: impact of non-lead based plumbing solders. *Toxicol. Environ. Chem.* 44, 11-20.
- Succop, P., Bornschein, R., Brown, K., Tseng, C.-Y., 1998. An empirical comparison of lead exposure pathway models. *Environ. Health Perspect.* 106 (Suppl. 6), 1577-1583.
- Sutherland, R.A., Day, J.P., Bussen, J.O., 2003. Lead concentrations, isotope ratios, and source apportionment in road deposited elements, Honolulu, Oahu, Hawaii. *Water Air Soil Pollut.* 142, 165-186.
- Sutton, P.M., Athanasoulis, M., Flessel, P., Guirguis, G., Haan, M., Schlag, R., et al., 1995. Lead levels in the household environment of children in three high-risk communities in California. *Environ. Res.* 68, 45-57.
- Tepper, L.B., Levin, L.S., 1975. A survey of air and population lead levels in selected American communities. *Environ. Qual. Saf. Suppl. II: Lead*, 152-195.
- Ter Haar, G., Aranow, R., 1974. New information on lead in dirt and dust as related to the childhood lead problem. *Environ. Health Perspect.* 7, 83-89.
- TerraGraphics Environmental Engineering, 2000. 1999 Populated Area Five Year Study Review, vol. II. 1999 Five Year Review Report: Bunker Hill Superfund Site, Table 2.3.
- Thomas, H.F., Elwood, P.C., Welsby St., E., Leger, A.S., 1979. Relationship of blood lead in women and children to domestic water lead. *Nature* 282, 712-713.
- Thompson, N.G., Sosnin, H.A., 1985. Corrosion of 50-50 tin-lead solder in household plumbing. *Welding J.* April, 20-24.
- Thomson, I., 1988. Metal content of soils and dusts. *Sci. Total Environ.* 75, 21-39.
- Torfs, K., Van Grieken, R., 1997. Chemical relations between atmospheric aerosols deposition and stone decay layers on historic buildings at the Mediterranean coast. *Atmos. Environ.* 31, 2179-2192.
- Treweek, G.P., Glicker, J., Chow, B., Sprinkler, M., 1985. Pilot-plant simulation of corrosion in domestic pipe materials. *J. Am. Water Works Assoc.* 77, 74-82.
- Turer, D., Maynard, J.B., Sansalone, J.J., 2001. Heavy metal contamination in soils of urban highways: comparison between runoff and soil concentrations at Cincinnati, Ohio. *Water Air Soil Pollut.* 132, 293-314.
- U.S. Centers for Disease Control, 1985. Preventing Lead Poisoning in Young Children. A Statement by the Centers for Disease Control. U.S. Department of Health and Human Services, Atlanta, GA.

- U.S. Centers for Disease Control, 1991. Preventing Lead Poisoning in Children. A Statement by the Centers for Disease Control. U.S. Department of Health and Human Services, Atlanta, GA.
- U.S. Centers for Disease Control, 2005. Preventing Lead Poisoning in Young Children. A Statement by the Centers for Disease Control. Department of Health and Human Services, Atlanta, GA.
- U.S. Department of Housing and Urban Development, 1995. Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing. Office of Lead Hazard Control, Washington, DC.
- U.S. Department of Housing and Urban Development, 1999. Requirements for Notification, Evaluation and Reduction of Lead-Based Paint Hazards in Federally-Owned Residential Property and Housing Receiving Federal Assistance. Final Rule.
- U.S. Department of Housing and Urban Development, 2001. National Survey of Lead and Allergens in Housing. Final Report.
- U.S. Environmental Protection Agency, 1971. Guidelines: Air Quality Surveillance Networks. Publication No. AP-98. Research Triangle Park, NC: Office of Air Programs. As cited in U.S. EPA, 1977, Ch. 4.
- U.S. Environmental Protection Agency, 1977. Air quality criteria for lead. Report No. EPA-600/8-77-017. Office of Research and Development, Washington, DC.
- U.S. Environmental Protection Agency, 1985. Occurrence of lead in drinking water, food, and air. Report by JRB Associates. Office of Drinking Water, Washington, DC. As cited in U.S. EPA, 1986b.
- U.S. Environmental Protection Agency, 1986a. Air Quality Criteria for Lead. 4 Vols. Report No. EPA/600/8-83/028bF. Environmental Criteria and Assessment Office, Washington, DC.
- U.S. Environmental Protection Agency, 1986b. Reducing lead in drinking water: a benefit analysis. Report No. EPA-230-09-86-019. Office of Policy Planning and Assessment, Washington, DC.
- U.S. Environmental Protection Agency, 1989. Risk assessment guidance for Superfund, vol. 1—Human Health Evaluation Manual (Part A). Report No. EPA/540/1-89/002. Office of Emergency and Remedial Response, Washington, DC.
- U.S. Environmental Protection Agency, 1994. Guidance Manual for the Integrated Exposure-Uptake Biokinetic Model for Lead in Children. EPA/540-R93/081. Office of Research and Development, Washington, DC.
- U.S. Environmental Protection Agency, 1995. National Air Quality and Emissions Trends Report, 1994. Report No. EPA 454/R-95-014. Office of Air and Radiation, Washington, DC.
- U.S. Environmental Protection Agency, 2001. Lead: Identification of Dangerous Levels of Lead. Final Rule. 40 CFR Part 745. FR 66: 1206–1240, January 5, 2001.
- U.S. Environmental Protection Agency, 2006. Air Quality Criteria for Lead, vol. 1. Report No. EPA/600/R-05/144aF. National Center for Environmental Assessment, Washington, DC.
- U.S. Environmental Protection Agency, 2007a. Air Toxics Data Archive. STAPPA/ALAPCO/U.S. EPA Air Toxics Monitoring Subcommittee. <<http://vista.cira.colostate.edu/atda/>> (accessed October 30, 2010).
- U.S. Environmental Protection Agency, 2007b. Lead: Human Exposure and Health Risk Assessments for Selected Case Studies, vol. 1. Human Exposure and Health Risk Assessments—Full Scale. Report No. EPA-452/R-07-014a. Office of Air Quality Planning and Standards, Research Triangle Park, NC.
- U.S. Environmental Protection Agency, 2008a. Child-Specific Exposure Factors Handbook. Report No. EPA/600/R-06/096F. National Center for Environmental Assessment, Washington, DC.

- Preventing Lead Poisoning in Children. A Statement by the Department of Health and Human Services, Atlanta, GA.
2005. Preventing Lead Poisoning in Young Children. Disease Control. Department of Health and Human Services.
- Urban Development, 1995. Guidelines for the Evaluation and Hazards in Housing. Office of Lead Hazard Control.
- Urban Development, 1999. Requirements for Notification. Lead-Based Paint Hazards in Federally-Owned Residential Federal Assistance. Final Rule.
- Urban Development, 2001. National Survey of Lead and Air Pollution.
- EPA, 1971. Guidelines: Air Quality Surveillance Networks. Triangle Park, NC: Office of Air Programs. As cited in EPA, 1977.
- EPA, 1977. Air quality criteria for lead. Report No. EPA-600/4-77-010. Urban Development, Washington, DC.
- EPA, 1985. Occurrence of lead in drinking water, food, and air. Office of Drinking Water, Washington, DC. As cited in EPA, 1986a.
- EPA, 1986a. Air Quality Criteria for Lead. 4 Vols. Report No. EPA-600/4-86-019. Office of Policy Planning and Assessment, Washington, DC.
- EPA, 1989. Risk assessment guidance for Superfund, vol. 1—Part A. Report No. EPA/540/1-89/002. Office of Research and Development, Washington, DC.
- EPA, 1994. Guidance Manual for the Integrated Exposure and Dose Assessment. EPA/540-R93/081. Office of Research and Development, Washington, DC.
- EPA, 1995. National Air Quality and Emissions Trends Report. EPA-454/R-95-014. Office of Air and Radiation, Washington, DC.
- EPA, 2001. Lead: Identification of Dangerous Levels of Lead. EPA-454/R-01-014. Office of Air and Radiation, Washington, DC. FR 66: 1206–1240, January 5, 2001.
- EPA, 2006. Air Quality Criteria for Lead, vol. 1. Report No. EPA-454/R-06-001. Office of Air and Radiation, Washington, DC.
- EPA, 2007a. Air Toxics Data Archive. STAPPA/ALAPCO/UTD. Triangle Park, NC. <<http://vista.cira.colostate.edu/atda/>>
- EPA, 2007b. Lead: Human Exposure and Health Risk Assessment. Case Studies, vol. 1. Human Exposure and Health Risk Assessment. Report No. EPA-452/R-07-014a. Office of Air Quality Planning, Triangle Park, NC.
- EPA, 2008a. Child-Specific Exposure Factors Handbook. EPA-452/R-08-001. Office of Air Quality Planning, Triangle Park, NC.
- EPA, 2008b. Office of the Administrator. National Ambient Air Quality Standards for Lead. Final Rule. Federal Register 73 FR 66964-67062, November 12, 2008.
- U.S. Food and Drug Administration, 1985. Market-basket survey: preliminary results for lead analysis. As cited in U.S. EPA, 1986a, Ch. 7.
- U.S. Food and Drug Administration, 2004. U.S. FDA Total Diet Study—Analytical Results. <<http://www.fda.gov/Food/FoodSafety/FoodContaminantsAdulteration/TotalDietStudy/ucm184293.htm>> (accessed November 3, 2010).
- von Lindern, I.H., Spalinger, S.M., Bero, B.N., Petrosyan, V., von Braun, M.C., 2003a. The influence of soil remediation on lead in house dust. *Sci. Total Environ.* 303, 59–78.
- von Lindern, I., Spalinger, S., Petrosyan, V., von Braun, M.C., 2003b. Assessing remedial effectiveness through the blood lead soil/dust relationship at the Bunker Hill site Superfund site in the Silver Valley of Idaho. *Sci. Total Environ.* 303, 139–170.
- Walter, S.D., Yankel, A.J., von Lindern, I.H., 1980. Age-specific risk factors for lead absorption in children. *Arch. Environ. Health* 35, 53–58.
- Water Research Centre, 1983. Lead in Drinking Water. Burke, T. (Ed.), Technical Report No. TR187. WRC Environmental Protection, Marlow, Bucks, United Kingdom.
- Welter, E., Calmano, W., Mangold, S., Tröger, L., 1999. Chemical speciation of heavy metals in soils by use of XAFS spectrometry and electron microscopic techniques. *Fresenius J. Anal. Chem.* 364, 238–244.
- World Health Organization, 1993. Guidelines for Drinking Water Quality. second ed. WHO, Geneva, Switzerland, vol. 1: Recommendations.
- World Health Organization, 1995. Environmental Health Criteria 165: Inorganic Lead. International Programme on Chemical Safety, Geneva, Switzerland.
- Worth, D., Matraga, A., Lieberman, M., Devas, E., Karalekas, P., Ryan, C., et al., 1981. Lead in drinking water: the contribution of household tap water to blood lead levels. In: Lynam, D.R., Piantinida, L.E., Cole, J.F. (Eds.), *Environmental Lead. Proceedings of the Second International Symposium on Environmental Lead Research*. Academic Press, New York, pp. 199–225.
- Yankel, A.J., von Lindern, I.H., Walter, S.D., 1977. The Silver Valley lead study: the relationship between childhood blood lead levels and environmental exposure. *J. Air Pollut. Control Assoc.* 27, 763–767.
- Yocum, J.E., 1982. Indoor–outdoor air quality relationships: a critical review. *J. Air Pollut. Control Assoc.* 32, 500–520.
- Young, T.M., Heeraman, D.A., Sirin, G., Ashbaugh, L.L., 2002. Resuspension of soil as a source of airborne lead near industrial facilities and highways. *Environ. Sci. Technol.* 36, 2484–2490.

Lead Exposure in Human Populations: Lead Intakes

7.1 INTRODUCTION

Chapters in this part of the book present and discuss lead exposure in human populations. Exposure here refers to actual, not potential, contact with lead in one or more environmental media by human populations. Furthermore, the term here also refers to both intake of media lead into various receiving body compartments and subsequent uptake into the human body. Other definitions of the term exist in the lead literature. For example, exposure is confined to lead intake into receiving body compartments and lead contact by an absorbing surface.

Four chapters comprise Part 2 of this monograph: Chapter 7 describes lead intakes in U.S. and other populations; Chapter 8 addresses Pb toxicokinetics and biological markers of human lead exposure; Chapter 9 presents modeling data reported for human lead exposures; and Chapter 10 describes the environmental epidemiology of human lead exposures.

Chapter 7 is the toxicological interface between lead in the external environment and lead in the human body; that is, it connects Chapters 6 and 8. Chapter 6 addressed lead levels in environmental media relevant to human contact. Chapter 8, the internal component of the interface, deals with lead disposition in the body. The latter describes the various kinetic and metabolic processes that govern Pb entry into, distribution within, and subsequent retention or excretion of internal lead burdens in exposed human populations. The older literature regarding lead did not always make clear distinctions between lead intake and lead uptake, which are two distinct processes in human lead exposures. Lead uptake deals with lead absorption into the bloodstream from receiving body compartments. In most cases, the distinction between the processes of intake and uptake are physiologically and toxicologically clear. Ingestion or inhalation of media-specific lead into those respective receiving body compartments occurs first, followed by absorption of some fraction of lead in those intakes into the bloodstream. In the case of

dermal contact, the distinction between the two steps is blurred since intake through skin is not fully distinct from uptake. With parenteral, e.g., intravenous, administration, the two merge into a single step.

Lead intake by inhalation occurs in two stages. First, some fraction of inhaled lead is retained and deposited in various compartments of the respiratory tract. The balance is exhaled. With inhalation, depositions in different parts of the respiratory tract yield different overall exposure results. Larger particles containing lead are deposited in the upper tract and undergo ciliary removal and are swallowed, passing into the gastrointestinal tract. The smallest particles containing lead are deposited in the pulmonary compartment. Sites for actual lead uptake into the bloodstream and the uptake characteristics for these different sites influence uptake rates.

TABLE 7.1 Illustrative Combinations of Pb Concentrations and Daily Environmental Media Intake Amounts Demonstrating Equivalent Intake Parameters^{a-c}

Environmental Medium	Media Pb Concentration (ppm or $\mu\text{g}/\text{m}^3$)	Daily Intake Mass (g) or Volume (m^3 , l)	Daily Pb Intake ($\mu\text{g}/\text{day}$)
Lead paint dust	5,000 (0.5%)	0.001 g	5
Lead paint dust	10,000 (1.0%)	0.001 g	10
Lead paint dust	25,000 (2.5%)	0.001 g	25
Lead in diet	0.0025	2,000 g	5
Lead in diet	0.005	2,000 g	10
Lead in diet	0.010	2,000 g	20
Lead in soil	500	0.010 g	5
Lead in soil	1,000	0.010 g	10
Lead in soil	2,000	0.010 g	20
Lead in tap water	0.005	1.0 l	5
Lead in tap water	0.010	1.0 l	10
Lead in tap water	0.025	1.0 l	25
Lead in air	1.0	5 m^3	5
Lead in air	2.0	5 m^3	10
Lead in air	5.0	5 m^3	25

^aMedia Pb levels for paint, soil, and tap water at or above regulatory levels.

^bIngested amounts typical for young children.

^cAir ventilation rate = 5 m^3 for young children.

between the two steps is blurred since intake is not distinct from uptake. With parenteral, e.g., intravenous, uptake and intake merge into a single step.

Uptake occurs in two stages. First, some fraction of the lead is deposited in various compartments of the respiratory tract. With inhalation, depositions in different compartments yield different overall exposure results. Larger amounts are deposited in the upper tract and undergo ciliary transport into the gastrointestinal tract. The smaller amounts are deposited in the pulmonary compartment, pass into the bloodstream and the uptake characteristics influence uptake rates.

Table 7.1 Concentrations of Pb and Daily Intake Amounts Demonstrating Equivalent Intake

Pb Concentration ($\mu\text{g}/\text{m}^3$)	Daily Intake Mass (g) or Volume (m^3 , l)	Daily Pb Intake ($\mu\text{g}/\text{day}$)
0.5%	0.001 g	5
(1.0%)	0.001 g	10
(2.5%)	0.001 g	25
	2,000 g	5
	2,000 g	10
	2,000 g	20
	0.010 g	5
	0.010 g	10
	0.010 g	20
	1.0 l	5
	1.0 l	10
	1.0 l	25
	5 m^3	5
	5 m^3	10
	5 m^3	25

0.5% water at or above regulatory levels.
children.
children.

Intake of lead or other substances in humans is typically indexed as daily intake. However, intakes scaled for different time frames have been employed in different settings over the years, e.g., weekly intakes (FAO/WHO, 1993) of Pb and other contaminants per body weight. This specifically applies to ingestion, inhalation, and, in some cases, dermal application. Daily lead intake into body compartments is the product of lead concentration in some medium and the mass (diet Pb) or volume (air Pb, water Pb) of lead-containing medium taken in daily. High levels of lead in an environmental medium can be quite toxic when ingested in relatively modest quantities daily.

On the other hand, low concentrations of lead can be of concern when large amounts of the lead-containing medium are ingested. The relationships are depicted in Table 7.1 where a high-lead medium such as lead paint dust ingested in small amounts can pose the same risk as low lead in the daily diet consumed at typical food intake levels. Table 7.1 shows that, because of either high Pb concentrations or high amounts of daily media mass intake, equivalent amounts of Pb can hypothetically enter receiving compartments daily regardless of the nature of the contact medium.

7.2 DAILY LEAD INTAKES BY U.S. AND OTHER POPULATIONS

Chapter 6 had numerous tabulations of lead concentrations in environmental media encountered by human populations in the United States and around the world. This chapter presents estimates for daily Pb intakes using Chapter 6 information combined with information on media-specific intake amounts. Intakes are provided for the major media-specific routes of exposure as they are available for various segments of human populations. Relevance of lead-containing environmental media differs across segments of populations, with given concentrations of lead in some contact medium translating into quite different degrees of lead exposure, depending on population and individual characteristics. For example, infants and toddlers ingest food at much higher rates on some body metric basis than older children or adults. Infants and toddlers ingest more of some particular Pb-contaminated environmental medium than do older individuals. A notable example is dust or soil by way of normal hand-mouth activity in this developmental age band. Dietary lead intakes differ with age as a function of type of dietary item, caloric requirements, etc. Groups of individuals with the same demographic characteristic but affected by different external factors such as climate and temperature will ingest different amounts of Pb-contaminated media, e.g., volumes of water.

A major determinant of lead intakes among human populations has been the period for which data were gathered. In past decades when lead levels were much higher than today in canned foods, in ambient air, in crops, and

in household painted surfaces, lead intake estimates were much higher. To the extent possible, available data for various periods are included in this chapter's tabulated intake estimates in order to show temporal changes in exposures. The reason is the same as for using older data in Chapter 6. Bone Pb in older subjects will reflect lead intakes from prior decades.

Lead-containing environmental media ingestion or inhalation rates are presented here for ambient air, diverse human diets, drinking water, and dust and/or soil. Dermal contact and potential dermal uptake into the bloodstream for the inorganic forms of environmental Pb encountered by nonoccupational populations are very low compared to the other routes of exposure, and this route is not addressed.

Various primary sources for estimating the exposure parameters associated with daily intake quantities of (lead-containing) media were used and are noted. A child exposure-specific source of parameter selection for media of interest was the U.S. EPA "Child-Specific Exposure Factors Handbook" (2008).

7.2.1 Daily Human Intakes of Ambient Air Lead

Daily intakes of lead through inhalation by human or experimental populations are the product of ventilation rate and air Pb concentration, further adjusted for lead deposition rates of the inhaled volumes in the respiratory tract. The ventilation rate, i.e., total volumes of inhaled air per unit time, typically 24 hours, can be determined by either direct measurement or via food-energy intakes. The latter may also be employed to arrive at the ratios of total energy expenditure to basal metabolic rate (BMR).

Layton (1993) carried out a series of studies with U.S. subjects of different ages for long-term and short-term inhalation rates estimated via various methodologies. Choice of methodology depended on the activity being evaluated. Daily long-term inhalation rates, which are the estimates most relevant to this chapter, were calculated from food-energy intakes. Table 7.2 presents the daily inhalation (ventilation) rates for children up to 18 years of age. Children under 9 years of age showed no gender-specific inhalation rates, but those 9 years and older showed a measurable difference favoring a higher rate for male children. Table 7.2 shows that inhalation rates for older male children were highest between 15 and 18 years, and for females, between 9 and 11 years. For those youngest children not identified by gender, the highest inhalation rates were calculated between the ages of 6 and 8.

Table 7.3 depicts estimates of long-term daily deposition rates of Pb in young children who varied in age and gender using available empirical data. Pb deposition rates increased with child's age in the range 1.4–3.2 $\mu\text{g Pb/day}$. Arcus-Arth and Blaisdell (2007) reported the statistical distributions of daily breathing rates for narrow age groups of infants and children.

surfaces, lead intake estimates were much higher. To obtain comparable data for various periods are included in this chapter in order to show temporal changes in lead intake estimates as for using older data in Chapter 6. Bone lead levels reflect lead intakes from prior decades.

Environmental media ingestion or inhalation rates are not air, diverse human diets, drinking water, and dust ingestion and potential dermal uptake into the bloodstream of environmental Pb encountered by nonoccupational exposure compared to the other routes of exposure, and this

procedures for estimating the exposure parameters associated with quantities of (lead-containing) media were used and EPA-specific source of parameter selection for media ingestion. EPA "Child-Specific Exposure Factors Handbook"

Intakes of Ambient Air Lead

Through inhalation by human or experimental populations, ventilation rate and air Pb concentration, further estimates of the inhaled volumes in the respiratory tract, i.e., total volumes of inhaled air per unit time, type of activity, determined by either direct measurement or via food-energy intake may also be employed to arrive at the ratios of inhaled air to basal metabolic rate (BMR).

Out of a series of studies with U.S. subjects of different ages, short-term inhalation rates estimated via various methodologies depended on the activity being evaluated. Inhalation rates, which are the estimates most relevant to estimating lead intake, are calculated from food-energy intakes. Table 7.2 presents inhalation rates for children up to 18 years of age. Table 7.2 shows that inhalation rates for older male children showed no gender-specific inhalation rates, while for females, between 9 and 18 years, showed a measurable difference favoring a higher inhalation rate. Table 7.2 shows that inhalation rates for older male children between 15 and 18 years, and for females, between 9 and 18 years, showed a measurable difference favoring a higher inhalation rate for males than for females. The highest inhalation rates were calculated between the ages of 6 and 8.

Estimates of long-term daily deposition rates of Pb in children, based on age and gender using available empirical data, are presented with child's age in the range 1.4–3.2 $\mu\text{g Pb/day}$. (2007) reported the statistical distributions of daily deposition rates for age groups of infants and children.

TABLE 7.2 Estimated Daily Long-Term Inhalation Rates for U.S. Children of Differing Ages^{a-c}

Group/Age (years)	Duration ^d	Mean Daily Inhalation Rate (m^3/day)
Children		
<1	1	4.5
1–2	2	6.8
3–5	3	8.3
6–8	3	19
Children—Males		
9–11	3	14
12–14	3	15
15–18	4	17
Children—Females		
9–11	3	13
12–14	3	12
15–18	4	12

^aAdapted from Layton (1993) and U.S. EPA (2008).

^bBased on food-energy intake rates from Layton (1993).

^cGender-specific data for children ≥ 9 years of age and adults.

^dNumber of years/cohort.

Tables 7.4 and 7.5 present the calculated daily inhalation rates and deposition rates for Pb at a time of maximum air Pb in the 1970s and then in 1994, when air Pb concentrations had essentially declined to their lowest levels. The pronounced distinctions in inhaled and deposited Pb in children at these two time points are striking, showing a 20-fold or more decline in deposition rates for urban areas. Again, the air Pb declines and associated inhaled and deposited Pb reflected the leaded gasoline ban in the United States and other industrialized nations. Daily inhaled Pb is deposited in various parts of the respiratory tract at a rate of 30% for children and adults (U.S. EPA, 1986, Ch. 10). Male children show a somewhat higher daily deposition rate than females. While the tables are for U.S. estimates, the values for air Pb outside the United States and those that are tabulated in Chapter 6 would show similar results.

Data given in Tables 7.2–7.4 are not expressed normalized to body weight or some other somatic metric. However, the nature of the

TABLE 7.3 Estimated Long-Term Daily Inhaled and Deposited Pb in U.S. Children of Various Ages for 1970–1974^a

Group/Age (years)	Daily Inhalation (m ³ /day) ^b	Daily Inhaled Pb (μg/day) ^c	Daily Deposited Pb (μg/day) ^d
Children			
<1	4.5	4.8	1.4
1–2	6.8	7.3	2.2
3–5	8.3	8.9	2.7
6–8	10	10.7	3.2
Males			
9–11	14	15.0	4.5
12–14	15	16.1	4.8
15–18	17	18.2	5.5
Females			
9–11	13	13.9	4.2
12–14	12	12.8	3.8
15–18	12	12.8	3.8

^aAverage of five urban U.S. arithmetic air Pb mean, 1970–1974, is given in Table 6.3: 1.07 μg/m³.^bAdapted from Layton (1993) and U.S. EPA (2008).^cAir Pb mean (μg/m³) × Daily Inhalation Rate (m³/day).^dDaily inhaled Pb (μg/day) × 0.3 (deposition rate).

methodology employed by Layton (1993), involving the dependence of air inhalation rates on food-energy intake rates, scaled total inhalation values in a manner that reflected body mass; consequently, when one adjusts these data for body weight, there is limited change across children's ages.

Adult inhalation rates for males and females generally cluster around 20 m³/day for individuals with typical occupational activity patterns in temperate climates. One can estimate the daily Pb inhalation and deposition rates for nonoccupational inhalation Pb exposures by using this value to arrive at values corresponding to those in Tables 7.3 and 7.4 for children.

7.2.2 Daily Human Intakes of Dietary Lead

Dietary lead concentrations relevant to human populations are presented in Chapter 6 and include periodic surveys from various agencies such as the U.S. FDA. This chapter has estimates of ingested daily amounts of dietary

Term Daily Inhaled and Deposited Pb in U.S. for 1970–1974^a

Inhalation	Daily Inhaled Pb ($\mu\text{g/day}$) ^c	Daily Deposited Pb ($\mu\text{g/day}$) ^d
	4.8	1.4
	7.3	2.2
	8.9	2.7
	10.7	3.2
	15.0	4.5
	16.1	4.8
	18.2	5.5
	13.9	4.2
	12.8	3.8
	12.8	3.8

^aUrban mean air Pb mean, 1970–1974, is given in Table 6.3: $1.07 \mu\text{g}/\text{m}^3$.
^bU.S. EPA (2008).
^cInhalation Rate (m^3/day).
^dDeposition rate).

Layton (1993), involving the dependence of air energy intake rates, scaled total inhalation values in body mass; consequently, when one adjusts these is limited change across children's ages. for males and females generally cluster around with typical occupational activity patterns in estimate the daily Pb inhalation and deposition rates Pb exposures by using this value to arrive at use in Tables 7.3 and 7.4 for children.

Intakes of Dietary Lead

relevant to human populations are presented in periodic surveys from various agencies such as the estimates of ingested daily amounts of dietary

TABLE 7.4 Estimated Daily Long-Term Inhaled and Deposited Pb in U.S. Children of Various Ages for 1994^a

Group/Age (years)	Daily Inhalation Rate (m^3/day) ^b	Daily Inhaled Pb ($\mu\text{g/day}$) ^c	Daily Deposited Pb ($\mu\text{g/day}$) ^d
Children			
<1	4.5	0.23	0.07
1–2	6.8	0.34	0.10
3–5	8.3	0.42	0.13
6–8	10	0.50	0.15
Males			
9–11	14	0.70	0.21
12–14	15	0.75	0.23
15–18	17	0.85	0.26
Females			
9–11	13	0.65	0.20
12–14	12	0.60	0.18
15–18	12	0.60	0.18

^aUrban mean air Pb for U.S. sites, U.S. EPA (1995), Table A-10.

^bAdapted from Layton (1993) and U.S. EPA (2008).

^cAir Pb mean ($\mu\text{g}/\text{m}^3$) \times Daily Inhalation Rate (m^3/day).

^dDaily Inhaled Pb ($\mu\text{g/day}$) \times 0.3 (deposition rate).

components and, based on these amounts and food group Pb levels given in the previous chapter, estimates of intakes of daily dietary Pb.

Human exposures to lead in the diet historically comprised a major fraction of overall lead exposures in the United States and elsewhere, especially for adults. However, in a number of instances, such as infant consumption of Pb in evaporated milk from lead-seamed cans, many children also sustained significant dietary Pb exposures. Dietary lead intakes, for purposes of this chapter, are separated from drinking beverages prepared from drinking water, but include foods cooked in tap water containing Pb.

Dietary components showed major reductions in Pb content over the last several decades, and Pb intakes through the diet obviously track these declines. Dietary Pb intake estimates in earlier decades were poorly recorded. Available data, however, clearly indicate that dietary Pb intakes during the era of leaded gasoline and lead-seamed cans for foods combined

TABLE 7.5 U.S. Daily Intakes of Food and Beverages During the Early 1980s^{a-c}

Food/Beverage Group	Child, 2 Years Old	14-16 Years Old		25-30 Years Old		60-65 Years Old	
		Male	Female	Male	Female	Male	Female
Meats/meat products	133	269	182	319	194	252	172
Food crops	282	528	386	518	390	532	437
Dairy	390	645	405	351	245	279	208
Canned foods	72	104	77	103	73	119	99
Canned juices	54	30	28	27	28	12	17
Frozen juices	65	75	53	73	66	61	72
Soda	65	274	232	315	228	85	78
Canned beer	0	17	0	318	51	116	18
Water	441	743	596	1,061	903	1,244	1,166
Total	1,502	2,685	1,959	3,086	2,178	2,700	2,267

^aAdapted from U.S. EPA (1986), and using summary data of Pennington (1983).^bUnits of g/day.^c"Water" category includes beverages such as tea, coffee, and powdered drinks.

to produce huge daily Pb intakes in diet because dietary components were contaminated by Pb.

Analogous to the approach for ambient air, this section first addresses estimates of the total amounts of diet ingested daily and components within the total dietary profiles for various groups within the U.S. and other populations. The increased uncertainty and variability in more generalized approaches for quantification of Pb in foodstuffs is amplified when approaching dietary intake amounts on a total or group-specific mass basis. For example, despite more public interest in "healthy" foods, a high per capita consumption rate of "fast" foods further complicates comparisons of historical dietary Pb intakes with those of recent years.

Food supplies of developed, industrialized countries in North America and Europe have centralized distribution systems and one would expect that the level of heterogeneity for lead distribution in dietary components would vary less across smaller subdivisions of nations than, say, ambient air Pb, dust or soil Pb, and tap water Pb. Variability and uncertainty remain

Intakes of Food and Beverages During the Early

Years	14–16 Years Old		25–30 Years Old		60–65 Years Old	
	Male	Female	Male	Female	Male	Female
1	269	182	319	194	252	172
2	528	386	518	390	532	437
3	645	405	351	245	279	208
4	104	77	103	73	119	99
5	30	28	27	28	12	17
6	75	53	73	66	61	72
7	274	232	315	228	85	78
8	17	0	318	51	116	18
9	743	596	1,061	903	1,244	1,166
10	2,685	1,959	3,086	2,178	2,700	2,267

Using summary data of Pennington (1983).

Includes such as tea, coffee, and powdered drinks.

Intakes in diet because dietary components were

For ambient air, this section first addresses estimates of diet ingested daily and components within the various groups within the U.S. and other populations. The variability in more generalized approaches for foodstuffs is amplified when approaching dietary intake on a group-specific mass basis. For example, despite the "healthy" foods, a high per capita consumption rate of food categories compares historical dietary Pb intakes

Developed, industrialized countries in North America and distribution systems and one would expect that for lead distribution in dietary components would be subdivisions of nations than, say, ambient air Pb or water Pb. Variability and uncertainty remain

nonetheless. First, there are socioeconomic components to diets within populations that translate to different amounts of Pb intakes and exposures.

Low-income subsets are more likely to produce more homegrown diet components in gardens. Groups of the population who rely on subsistence fishing and harvest catches where aquatic lead contamination is likely or of concern will ingest amounts of the element significantly above mean population intakes. Ethnic food preferences can also raise the likelihood of higher Pb content in certain imported canned foods.

The principal sources of systemic assessments of dietary intakes of food groups in the United States are the USDA's NFCS and the USDA Continuing Survey of Food Intakes by Individuals (CSFII) (U.S. Department of Agriculture, 1990). The NFCS databases include figures for 1987/1988. The CSFII values are for two more recent periods, 1989–1991 and 1994–1996, and were used for a number of estimated intakes. The U.S. EPA exposure factors handbook for children (U.S. EPA, 2008) was also used. Tables 7.5–7.8 present estimates of daily amounts of food groups ingested in various U.S. population age groups. Values are in two forms, as daily weights per person and/or dietary amounts on a body weight basis. Table 7.5 shows that a typical 2-year-old child ingests around 1 kg of various food groups and about 440 ml of water. With age, both food and water amounts consumed daily increase, peaking in the 25- to 30-year-old U.S. male at around 3.1 kg and in the U.S. female at 60–65 years of age.

Table 7.6 shows daily amounts of food ingested by U.S. children in the overall age range of <1 year old to 19 years old. The teenaged band comprises the highest total daily intake group, showing 200 g more on average than infants. However, when intakes are adjusted to body weight, the highest intake rate is in early infants and least in the 12 to 19-year-olds, a factor of about five higher in the infants. Distribution of ingested daily amounts of food into food group categories as a function of body weight is tabulated in Table 7.7. While absolute daily intakes differ across ages, all children consume the same food group in proportional amounts in terms of mean and 95th percentile estimates. Dairy products rank highest across all ages in terms of daily intake while the "fish" category ranks lowest. Table 7.8 presents the amounts of daily food group intake for the "homegrown" food items. The first part gives the fraction of children consuming homegrown foods while the remainder tabulates mean and 95th percentile intakes as a function of body weight.

Daily intakes of dietary Pb vary with age and gender. U.S. dietary Pb intakes are presented in Table 7.9 for the early 1980s. This period reflected some persisting impact of leaded gasoline use in the nation, based on data presented in Chapter 6 for food group lead concentrations over the decades. Teenagers and young adults consumed more Pb on a total daily intake basis than young children or the elderly, as shown in Table 7.9. Gender differences in daily U.S. dietary Pb intakes are seen in the teen, young adult, and older

TABLE 7.6 U.S. Per Capita Total Diet Intakes for Children at Different Ages^{a-c}

Child Age Band (years)	Mean	95th Percentile
Diet intake, g/day, as consumed		
<1	1,000	1,800
1-2	1,100	1,800
3-5	1,000	1,700
6-11	1,100	1,900
12-19	1,200	2,300
Diet intake, g/kg-day, as consumed		
<1	140	240
1-2	84	150
3-5	55	100
6-11	36	69
12-19	26	40

^aAdapted from U.S. EPA (2008) based on 1994/1996 USDA CSFII.^bIntake amounts as consumed.^cMean/95th percentile values.**TABLE 7.7** U.S. Per Capita Total Intakes (Mean/95th Percentile) of Different Consumer Food Categories for Children of Various Ages^{a-c}

Food Group	Children's Age Band (years)				
	<1	1-2	3-5	6-11	12-19
Meat	1.1/5.9	4.4/10.2	4.1/9.4	2.9/6.8	2.2/4.9
Fish	0.1/0.5	0.4/1.8	0.3/1.7	0.3/1.4	0.2/1.1
Vegetables	6.9/24.2	9.5/23.3	7.3/18.3	5.3/13.5	4.0/9.3
Grains	4.1/20.2	11.2/24.7	10.3/21.1	7.2/15.6	4.4/9.7
Dairy	111/235	37.5/90.2	20.9/48.8	13.9/33.5	6.1/17.8
Fruits	13.2/41.2	19.3/53.9	11.0/32.7	5.4/18.0	2.8/11.0

^aAdapted from U.S. EPA (2008) and using USDA CSFII 1994-1996 data.^bg/kg-day food group.^cGeneral population members, <1 to 19 years of age.

Total Diet Intakes for Children at Different

	Mean	95th Percentile
ed		
	1,000	1,800
	1,100	1,800
	1,000	1,700
	1,100	1,900
	1,200	2,300
umed		
	140	240
	84	150
	55	100
	36	69
	26	40

based on 1994/1996 USDA CSFII.

Total Intakes (Mean/95th Percentile) of
Categories for Children of Various Ages^{a-c}

Children's Age Band (years)			
1-2	3-5	6-11	12-19
4.4/10.2	4.1/9.4	2.9/6.8	2.2/4.9
0.4/1.8	0.3/1.7	0.3/1.4	0.2/1.1
9.5/23.3	7.3/18.3	5.3/13.5	4.0/9.3
11.2/24.7	10.3/21.1	7.2/15.6	4.4/9.7
37.5/90.2	20.9/48.8	13.9/33.5	6.1/17.8
19.3/53.9	11.0/32.7	5.4/18.0	2.8/11.0

and using USDA CSFII 1994-1996 data.

1 to 19 years of age.

TABLE 7.8 Mean U.S. Consumption of Homegrown Foods from Different Food Groups by Children of Various Ages^{a-c}

Food Group	% Children Consuming by Age (years)				Mean and 95th Percentile by Age (years)			
	1-2	3-5	6-11	12-19	1-2	3-5	6-11	12-19
Homegrown fruits	6.3	6.8	6.3	5.8	8.7/61	4.1/8.9	3.6/16	1.9/8.3
Homegrown vegetables	16.7	15.2	18.1	16.1	5.2/19.6	2.5/7.7	2.0/6.2	1.5/6.0
Home-produced meats	4.8	4.9	6.4	6.2	3.7/10	3.6/9.1	3.7/14	1.7/4.3
Home-caught fish	1.4	1.8	2.3	1.7	-	-	2.8/7.1	1.5/4.7

^aAdapted from U.S. EPA (2008), using USDA NCFS for 1987-1988.^bg/kg-day.^cInsufficient data for groups not indicated.TABLE 7.9 Total Pb Intake ($\mu\text{g/day}$), by Age and Sex, of Food and Beverages in the Early 1980s^{a-c}

Food Category	Child, 2 Years Old	14-16 Years Old		25-30 Years Old		60-65 Years Old	
		Male	Female	Male	Female	Male	Female
Meat/meat products	3.4	7.4	4.8	7.4	5.0	5.4	4.0
Food crops	5.5	11.7	8.1	11.3	7.9	9.6	7.8
Dairy	2.8	5.4	3.5	3.4	2.5	3.1	2.3
Canned foods	7.3	11.8	8.1	12.0	8.8	14.4	11.6
Canned juices	2.7	1.5	1.4	1.4	4.4	0.6	0.9
Frozen juices	0.5	0.7	0.5	0.7	0.6	0.5	0.7
Soda	0.7	3.0	2.3	2.9	2.1	0.9	0.9
Canned beer	0.0	0.1	0.0	2.5	0.7	1.0	0.3
Water	2.1	3.2	2.5	3.6	3.0	4.2	3.9
Total	25.0	44.8	31.2	45.2	32.0	39.7	42.4

^aAdapted from U.S. EPA (1986) and using data of Pennington (1983).^bUnits of $\mu\text{g/day}$.^c"Water" category includes coffee, tea, and powdered drinks.

adult groups, with males ingesting more Pb. No gender difference was seen with infants or toddlers.

U.S. population daily diet Pb intake declines with the phase out of leaded gasoline and lead seams in food or beverage cans are evident from the national data. Chapter 6 noted Pb concentration declines in various food groups from the 1970s to more recent years. In terms of total daily Pb intakes, a marked decline has been observed for all age and gender groups. Adams (1991) reported that for the relatively short period from 1982 to 1988, a period associated with reductions in leaded gasoline and lead-seamed can use, toddlers showed about a fivefold reduction in daily intakes from 25 to 5 $\mu\text{g}/\text{day}$. A similar relative reduction was seen with adult women from 36 to 8 $\mu\text{g}/\text{day}$.

Egan et al. (2002) reported lower daily U.S. diet Pb intakes into the 1990s. The 1994–1996 period showed daily dietary Pb intake ranges for (1) infants up to 11 months, (2) 2 year olds, and (3) older children or adults as 0.8–5.7, 2.4–10.1, and 4–19 $\mu\text{g}/\text{day}$, respectively. For 1982–1984, corresponding figures for the above age groups were (mean): 17, 23, and 29–41 $\mu\text{g}/\text{day}$. The 2004 U.S. FDA survey noted in Chapter 6 recorded virtually all categories of food groups for U.S. population segments as being below the measurement detection limit. Consequently, any current estimates of daily diet Pb would reflect statistically projected figures with considerable uncertainty. Suffice it to say that today's figures would likely be lower than those noted by Egan et al. (2002) for the mid-1990s period.

The trend of declining Pb in dietary daily intakes is readily apparent in U.S. EPA figures selected as exposure input values for dietary Pb to the agency's Integrated Exposure-Uptake Biokinetic Model for estimating PbB values in children (U.S. EPA, 2009). Table 7.10 summarizes Pb intakes for 12-month age bands in children up to 84 months of age. In infants of 12–23 months old, Pb intake declined about 65% between pre-1991 data and 1995–2003 data. Similar declines were seen in other age bands.

International daily Pb intake estimates for diet track a similar array of older values as in the United States and were summarized for the 1980s by Galal-Gorchev (1991). Table 7.11 summarizes the international picture for daily diet Pb intakes by children on the basis of body weight for nine countries representing various locales and demographic profiles. The U.S. weekly and daily diet Pb intakes adjusted to body weight were the lowest, 3.1 $\mu\text{g}/\text{week}$ and 0.4 $\mu\text{g}/\text{day}$, respectively. Poland ranked highest, at 28 $\mu\text{g}/\text{week}$ (4 $\mu\text{g}/\text{day}$). Corresponding figures for adults are presented in Table 7.12. India, Italy, and Cuba showed the highest diet Pb intakes on the basis of body weight, with the United States showing mean data (rounding) of <0.02 .

Some subsets of human populations have relatively limited dietary Pb sources. Nursing infants, for example, will ingest any Pb passing into breast milk from the nursing mother while bottle-fed infants can consume any tap

ingesting more Pb. No gender difference was seen

diet Pb intake declines with the phase out of leaded
s in food or beverage cans are evident from the
5 noted Pb concentration declines in various food
to more recent years. In terms of total daily Pb
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ates showing mean data (rounding) of <0.02 .

an populations have relatively limited dietary Pb
for example, will ingest any Pb passing into breast
other while bottle-fed infants can consume any tap

TABLE 7.10 Total Pb Intake ($\mu\text{g/day}$) by Child Age Band Based on Various U.S. EPA Updatings of Dietary Pb^{a-c}

Age Band (months)	Diet Pb Intake		
	Pre-1991	1991–1999 TDS Data	1995–2003 Data
0–11	5.5	3.2	2.3
12–23	5.8	2.6	2.0
24–35	6.5	2.9	2.1
36–47	6.2	2.7	2.0
48–59	6.0	2.6	2.0
60–71	6.3	2.7	2.1
72–84	7.0	3.0	2.2

^aBased on U.S. EPA (1994) model manual diet Pb estimates for 1980s.

^bBased on U.S. FDA Total Diet Study estimates: 1991–1999 and 1995–2003.

^cTotal age band, 0–84 months.

TABLE 7.11 Daily Pb Intakes in Child Population for Various Countries Surveyed in the UNEP/GEMS Program^{a-c}

Country	Total Weekly Pb Intake ($\mu\text{g/kg}$ body weight)	Total Daily Pb Intake ($\mu\text{g/kg}$ body weight)
Poland	27.7	4.0
Germany ^d	26.9	3.8
Hungary	25.8	3.7
Canada	17.6	2.5
Sweden	15.1	2.2
Philippines	12.6	1.8
United Kingdom	11.8	1.7
Cuba	7.3	1.0
United States	3.1	0.4

^aAdapted and estimated from values in Galal-Gorchev (1991) to daily intakes.

^bInfants and older children up to age 12, 1980–1988.

^c $\mu\text{g/kg}$ body weight.

^dPre-reunification figures for Federal Republic of Germany.

TABLE 7.12 Daily Pb Intakes in Adult Population for Various Countries Surveyed in the UNEP/GEMS Program^{a-c}

Country	Total Weekly Pb Intake ($\mu\text{g/kg}$ body weight)	Total Daily Pb Intake ($\mu\text{g/kg}$ body weight)
India	60.0	8.6
Italy	59.3	8.5
Cuba	63.3	9.0
Germany ^d	26.7	3.8
France	19.6	2.8
Poland	18.3	2.6
Japan	9.8	1.4
United Kingdom	7.2	1.0
Canada	5.7	0.8
Sweden	2.6	0.4
United States	0.02	0.0 ^e

^aAdapted and estimated from values in Galal-Gorchev (1991) for weekly intakes.

^bAdults for the period 1980–1988.

^c $\mu\text{g/kg}$ body weight.

^dPre-reunification figures for the Federal Republic of Germany.

^eRounding.

water Pb in formula mixtures. It is not now possible to provide an accurate estimate of daily Pb intakes by nursing. Earlier literature indicated such Pb exposures for infants might be of some significance, but analytical method sensitivity and persisting sample lead contamination have been problematic. More recent and comparatively more reliable measurement data reported by Gulson et al. (2003) suggest a daily intake figure for this pathway of about $0.8 \mu\text{g/day}$. The corresponding formula-based Pb intake is on the order of $1\text{--}2 \mu\text{g/day}$.

The marked decline in dietary Pb daily intakes from the 1970s to the present has produced a marked shift downward in the relative fractional contribution of diet Pb to total daily Pb intakes. For example, Bolger et al. (1991) estimated that the percentage contribution to total Pb intake in 2 year olds attributable to their diet declined from 47% in 1986 to 16% in 1990. This threefold decline occurred with major declines in leaded gasoline and lead-seamed food can use but relatively little decline in other major Pb pathways for children, e.g., soil Pb.

Intakes in Adult Population for Various Countries
GEMS Program^{a-c}

Total Weekly Pb Intake ($\mu\text{g/kg}$ body weight)	Total Daily Pb Intake ($\mu\text{g/kg}$ body weight)
50.0	8.6
59.3	8.5
53.3	9.0
26.7	3.8
9.6	2.8
8.3	2.6
9.8	1.4
7.2	1.0
5.7	0.8
2.6	0.4
0.02	0.0 ^e

Intakes in Galal-Gorchev (1991) for weekly intakes.
88.

Federal Republic of Germany.

ures. It is not now possible to provide an accurate
es by nursing. Earlier literature indicated such Pb
ht be of some significance, but analytical method
sample lead contamination have been problematic.
tively more reliable measurement data reported by
est a daily intake figure for this pathway of about
nding formula-based Pb intake is on the order of
n dietary Pb daily intakes from the 1970s to the
arked shift downward in the relative fractional con-
total daily Pb intakes. For example, Bolger et al.
percentage contribution to total Pb intake in 2 year
diet declined from 47% in 1986 to 16% in 1990.
ccurred with major declines in leaded gasoline and
but relatively little decline in other major Pb path-
l Pb.

TABLE 7.13 Estimated Direct Daily and Indirect Total Water Intakes by
Water Source for the U.S. Population^{a-d}

Water Source	Daily Water Intake (ml/day)		
	Mean	90th Percentile	95th Percentile
Community supply	927	2,016	2,544
Bottled water	161	591	1,036
Other sources	128	343	1,007
Missing sources	16	—	—
All sources	1,232	2,341	2,908

^aSources: Data from USDA CSFII (1998) and U.S. EPA, 2008.

^bDirect intakes = water consumed directly; indirect intakes = water used in food preparation.

^cBased on two nonconsecutive days of reporting, N = 15,303.

^dFor all ages in the population.

7.2.3 Daily Human Intakes of Drinking Water Lead

Various U.S. and other surveys of drinking water consumption rates differ considerably depending on what subdivisions of total fluid volume intakes are included in the various totals being reported. Some deal solely with daily direct water intake volumes while others include water quantities consumed directly and volumes used in daily food preparation. Still other surveys include bottled water or water volumes used for beverages as well as tap water volumes or include just community water sources in the intake tallies. The various tabulations presented in this section note that these qualifications were feasible.

Consumption volumes differ across age and gender in human populations, reflecting factors such as climate, physical exertion, and body mass. It should be noted, nonetheless, that it is the variability of Pb content that is the larger parameter contributing to both variability and uncertainty compared to quantities of water drunk daily. For example, levels of Pb in drinking water, especially in homes with lead-soldered plumbing or lead service lines, can vary by an order of magnitude or even more, while the range of imbibed water volumes will be considerably less.

Table 7.13 tabulates estimates of total (direct and indirect) water intakes as a function of water source for all ages in the U.S. population as provided by surveys done by the U.S. EPA (2008) and the USDA CSFII (1998) survey programs. The daily intake mean volumes for community, bottled water, other sources, and unknown sources are 927, 161, 128, and 16 ml, respectively, producing a total of 1,232 ml. The corresponding 90th and 95th

TABLE 7.14 Estimated Daily Direct and Indirect Community Water Intakes by Children's Age Category^{a-e}

Age Band (years)	Sample N	Mean	90th Percentile	95th Percentile
Intake (ml/day)				
0.5-0.9	160	412	884	1,101
1-3	1,834	313	691	942
4-6	1,203	420	917	1,165
7-10	943	453	978	1,219
15-19	825	760	1,610	2,062
Intake (ml/kg-day)				
0.5-0.9	153	45	103	122
1-3	1,752	23	51	67
4-6	1,103	21	44	64
7-10	879	15	32	39
15-19	816	12	25	32

^aAdapted from U.S. EPA (2008) and data from USDA-CSFII (1998).^bDirect intakes = water consumed directly from the tap; indirect intakes = water used in food preparation.^cBased on two nonconsecutive days of sampling.^dSample size varies with age band.^eReported as total daily intake/person or daily intake per kg body weight.

percentile values are 2,016 and 2,544 ml for community water and 591 and 1,036 ml for bottled water. The table shows that community water supplies, provided in large measure by public water treatment facilities, constitute the main source of the typical U.S. resident having public supply access. In terms of mean allocation of water volume intakes as a function of water sources, community supplies comprise about 75% of total water daily intake. For the 95th percentile of source-based water intakes, the corresponding figure is 87%.

Table 7.14 presents daily water intake estimates as a function of age within the childhood group, calculated both as total volumes per day and as a function of body weight. The source of water in these estimates is the community water system in the survey communities. Five age bands for children, along with their respective daily water intake volumes, are given in terms of total daily intakes or daily intakes on the basis of body weight. Total water intakes are highest in children of 15-19 years of age, 760 ml, and least for

Direct and Indirect Community Water Category^{a-e}

Mean	90th Percentile	95th Percentile
412	884	1,101
313	691	942
420	917	1,165
453	978	1,219
760	1,610	2,062
45	103	122
23	51	67
21	44	64
15	32	39
12	25	32

data from USDA-CSFII (1998).

data from USDA-CSFII (1998).
 directly from the tap; indirect intakes = water used in food.

of sampling.

on or daily intake per kg body weight.

and 2,544 ml for community water and 591 and 1,000 ml for private wells. The table shows that community water supplies, by public water treatment facilities, constitute the largest source of water for U.S. resident having public supply access. In terms of water volume intakes as a function of water source, public water supplies comprise about 75% of total water daily intake. When comparing source-based water intakes, the corresponding

ily water intake estimates as a function of age, calculated both as total volumes per day and as The source of water in these estimates is the com- survey communities. Five age bands for children, daily water intake volumes, are given in terms of intakes on the basis of body weight. Total water dren of 15-19 years of age, 760 ml, and least for

TABLE 7.15 Tap Water Daily Intake Rates for Women of Childbearing Age (15–49 years)^{a,b}

Reproductive Status	Mean	90th Percentile	95th Percentile
Daily intake, person (ml/day)			
Control	1,157	1,983	2,310
Pregnant	1,189	2,191	2,424
Lactating	1,310	1,945	2,191
Daily intake, person (ml/kg-day)			
Control	19.1	33.1	39.1
Pregnant	18.3	34.5	39.6
Lactating	21.4	35.1	37.4

^aAdapted from U.S. EPA (2008).

^bTap water fraction of daily fluid intake: control, 57.2%; pregnant, 54.1%; lactating, 57.0%.

those 1-3 years of age. However, on the basis of body weight, the values are largely reversed. Similar relative rankings were found at the 90th and 95th percentiles.

Table 7.15 presents water volume intakes among women of childbearing age, pregnant women, and those who are lactating. Such intake estimates provide a measure of potential lead exposure risks to the fetus and the newborn when the concentrations of Pb in these water sources are known. In terms of total water daily consumption, the volumes are similar in all three categories for mean (1,157–1,310 ml/day) or higher percentile consumption rates.

The global figure for daily drinking water Pb intake reported from the GEMS/Food/UNEP program by Galal-Gorchev (1991) is 40 $\mu\text{g/day}$. This estimate is based on an international consumption volume across age groups, gender, and nationalities of 2,000 ml and a global average water concentration of 0.02 $\mu\text{g/ml}$ or 20 $\mu\text{g Pb/l}$ water. Daily tap water Pb consumption rates have varied with time in U.S. populations of varying age. For the early 1980s, prior to controls on lead content of plumbing materials and plumbing practices (banning use of 50–50 lead–tin solder), total water Pb intakes for various ages were 2.1 $\mu\text{g/day}$, 2-year-olds; 3.2 and 2.5 $\mu\text{g/day}$, 14- to 16-year-old males and females, respectively; 3.6 and 3.0 $\mu\text{g/day}$, 25- to 30-year-old males and females, respectively; 4.2 and 3.9 $\mu\text{g/day}$, 60- to 65-year-old males and females, respectively.

Estimates of daily water Pb intakes can be readily calculated for more recent times with data for water Pb levels for typical communities (U.S.

EPA, 2006) in combination with daily consumption volumes recorded in tables presented in this chapter (Table 7.14). Table 7.16 tabulates estimated daily water Pb intakes for indicated communities and residents of different ages in those communities. The cities indicated in the table were recorded with exceedances of the current drinking water lead action level at the tap, a level of 15 $\mu\text{g/l}$ at the 90th percentile of distributions in survey results for these communities. These cities, therefore, represent more of a worst case scenario for U.S. urban community water supplies.

Table 7.16 presents data for two tap water surveys, in 1993 and some more recent survey results over the years 2001–2003 depending on the individual city. Older children 15–19 years old show the highest tap water lead intakes for both the 1993 and the more recent surveys, since they consume the highest volume of tap water among the given childhood age bands (0.76 l). Of the city tap water Pb levels recorded for 1993, Philadelphia, PA, had the highest intakes across the board, and was the highest among the 18 cities surveyed with action level exceedances. In Philadelphia, children of 15–19 years old experienced significant potential lead exposures from tap water, depending on household practices such as flushing or not flushing standing water from household plumbing. By contrast, during 2001–2003 this city's tap water Pb values were significantly lower and, consequently, children residing in this community had relatively lower intakes.

The tap water intakes for those 18 cities with action level exceedances for 2001–2003 were, overall, considerably below what they were in 1993. These reductions reflected a variety of water treatment and other reduction strategies implemented post-1993. Nonetheless, some communities, such as Washington, DC, still have the potential for tap water Pb exposures.

7.2.4 Daily Human Intakes of Soil Lead

Soil Pb intakes by segments of various populations can pose significant potential risks of exposures. However, the nature of soil Pb exposures are such that they present higher risks to sensitive groups such as young children than they do for adults. The reason for this is simply that the young child orally explores his/her physical environment and typically engages in various hand-to-mouth activities that include ingestion of varying amounts of soil. Older literature labeled all such normal hand-to-mouth activity as a "pica" behavior but that label is now reserved for "excessive" ingestion of nonfood materials. However, no clear clinical definition of, or criteria for, this behavior exists. Some literature has defined the behavior in statistical terms, e.g., some upper percentile of a distribution of observed daily ingestion rates within some study population. For discussing the broad topic of soil ingestion through normal child activity, it is probably best to dispense with the term and confine it to extreme intakes, e.g., gram quantities.

ation with daily consumption volumes recorded in chapter (Table 7.14). Table 7.16 tabulates estimated for indicated communities and residents of different ies. The cities indicated in the table were recorded current drinking water lead action level at the tap, a 90th percentile of distributions in survey results for se cities, therefore, represent more of a worst case community water supplies.

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Intakes of Soil Lead

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TABLE 7.16 Estimated Mean Daily Tap Water Pb Intakes for Children in the Indicated U.S. Communities for the Indicated Time Periods and Child Ages^{a,d}

Community	Period									
	1993					2001–2003				
	0.5–0.9 Years	1–3 Years	4–6 Years	7–10 Years	15–19 Years	0.5–0.9 Years	1–3 Years	4–6 Years	7–10 Years	15–19 Years
Bayonne, NJ	7	6	8	8	14	7	6	8	8	14
Cedar Rapids, IA	33	25	34	36	61	2	2	3	3	5
Chicago, IL	4	3	4	5	8	3	2	3	3	5
Columbia, SC	16	12	17	18	30	2	2	3	3	5
Columbus, OH	6	5	6	7	11	0	0	0	0	1
Detroit, MI	9	7	9	9	16	5	4	5	5	9
Galveston, TX	7	6	8	8	14	1	1	1	1	2
Miami Beach, FL	11	8	11	12	21	3	2	3	4	6
Minneapolis, MN	8	6	8	9	14	2	2	3	3	5
Philadelphia, PA	132	100	135	145	245	5	4	5	6	10
Phoenix, AZ	8	6	8	9	14	0	0	0	0	1
Portland, OR	17	13	17	18	31	3	2	3	4	6

(Continued)

TABLE 7.16 Estimated Mean Daily Tap Water Pb Intakes for Children in the Indicated U.S. Communities for the Indicated Time Periods and Child Ages^{a-d}—(cont.)

Community	Period									
	1993					2001–2003				
	0.5–0.9 Years	1–3 Years	4–6 Years	7–10 Years	15–19 Years	0.5–0.9 Years	1–3 Years	4–6 Years	7–10 Years	15–19 Years
Richmond, VA	7	5	7	7	12	2	1	2	2	3
St. Paul, MN	22	17	23	24	41	0	0	0	0	1
Syracuse, NY	21	16	21	23	38	10	8	11	11	19
Tacoma, WA	13	10	13	14	24	5	4	5	5	9
Yonkers, NY	28	21	29	31	52	7	6	8	8	14
Washington, DC	7	6	8	8	14	26	20	26	28	48

^aCommunities noted in U.S. EPA (2006: Table 3.11) as exceeding EPA Pb action levels.

^b90th percentile Pb levels ($\mu\text{g/l}$) for 1993 and most recent monitoring periods: 2001–2003.

^cVolumes of water consumed at different ages by children as given in Table 7.13. Excludes bottled water and minor categories.

^dMethods for tap water testing referenced in U.S. EPA (2006).

Community	0.5-0.9 Years	1-3 Years	4-6 Years	7-10 Years	15-19 Years	0.5-0.9 Years	1-3 Years	4-6 Years	7-10 Years	15-19 Years
Richmond, VA	7	5	7	7	12	2	1	2	2	3
St. Paul, MN	22	17	23	24	41	0	0	0	0	1
Syracuse, NY	21	16	21	23	38	10	8	11	11	19
Tacoma, WA	13	10	13	14	24	5	4	5	5	9
Yonkers, NY	28	21	29	31	52	7	6	8	8	14
Washington, DC	7	6	8	8	14	26	20	26	28	48

^aCommunities noted in U.S. EPA (2006: Table 3.11) as exceeding EPA Pb action levels.

^b90th percentile Pb levels ($\mu\text{g/l}$) for 1993 and most recent monitoring periods: 2001-2003.

^cVolumes of water consumed at different ages by children as given in Table 7.13. Excludes bottled water and minor categories.

^dMethods for tap water testing referenced in U.S. EPA (2006).

Two parameters determine daily soil Pb intake, much the same way they do with other environmental media. They are the soil Pb concentration and the daily amounts of soil ingested. Daily ingested amounts of soils vary in children, but the range of variation is dwarfed by the huge range of soil Pb concentrations the children may encounter. The latter reflect all of the contamination histories of specific soils encountered by children, including pathways for contamination such as atmospheric Pb fallout onto yard soils, weathering or chalking of exterior lead paints onto adjacent soils, and Pb-bearing waters runoff onto adjacent soils. While ingested amounts across individuals from early childhood through adulthood will typically lie within an order of magnitude, 20-200 mg, Pb concentrations can range from background, uncontaminated levels in the range of 20-50 ppm or so to seriously contaminated soils having lead content at percentage levels.

The role of soil Pb exposures in human Pb toxicity is of more concern for young children and has prompted various studies of daily ingestion rates for soils containing Pb or other contaminants. Ethical considerations preclude feeding Pb-laced soils to children so typical methods in the relevant literature involve using adult healthy volunteers or doing excretion studies for soil Pb ingested in normal play activities. Both these approaches require elemental "tracers" which are ingested by children along with various soils but are assumed to have virtually no absorption themselves. Typical studies among these approaches are by Binder et al. (1986), Calabrese and Stanek (1995), Calabrese et al. (1989), Calabrese et al. (1991), Calabrese et al. (1997), Clausen et al. (1987), Davis et al. (1990), Stanek and Calabrese (1995a,b), and Van Wijnen et al. (1990).

Table 7.17 provides summary information derived from the above reports. Note that the range of intake amounts of soil not only varies across investigators' reports but also within studies for different tracers used to monitor intake-excretion balances. Mean daily ingestion values across studies for soil alone ranged from 66 to 271 mg/day, with the upper percentiles well over 1,000 mg/day. Stanek and Calabrese (1995a,b) saw variation with tracer of daily ingestion rates by children in the studies showing a range of 85-218 mg/day based on four different geochemical tracers. Some of the studies, e.g., those of Calabrese et al. (1989), Calabrese et al. (1997), and Davis et al. (1990), reported intake data for dust and soil combined.

The studies cited above typically reported intakes over limited single points in time, leaving open the question of stability of the results. Stanek and Calabrese (1995a) extended the utility of these studies by carrying out estimated distributions of individual mean daily soil intake rates projected over 365 days. The median value for this estimating exercise was 75 mg/day, while the 90th and 95th percentiles of daily ingestion were 1,190 and 1,751 mg/day, respectively. The range was 1-2,268 mg/day.

Few studies of daily soil ingestion actually precisely differentiated soil from either interior or exterior dusts. Children ingest dusts in sufficient

TABLE 7.17 Soil Lead Intake Rates for Children Reported in Indicated Tracer Studies^{a-c}

Mean					Upper Percentile (mg/day)				References
Al	Si	AIR ^d	Ti	Y	Al	Si	Ti	Y	
181	184				584	578			Binder et al. (1986)
230		129							Clausing et al. (1987)
39	82		246						Davis et al. (1990)
65 ^e	160 ^e		268						Davis et al. (1990)
153	154		218	85	223	276	1,432	106	Calabrese et al. (1989)
154 ^e	483 ^e		170 ^e	65 ^c	478 ^c	653 ^c	1,059 ^e	159 ^c	Calabrese et al. (1989)
122	139		271	165	254	224	279	114	Stanek and Calabrese (1995a)
133 ^f					217 ^f				Stanek and Calabrese (1995a)
69-120									Van Wijnen et al. (1990)
66 ^f					280 ^f				Calabrese et al. (1997)
196 ^e					994 ^c				Calabrese et al. (1997)

^aAdapted from U.S. EPA (2008) and data contained in the indicated studies.^b"Upper percentile" as indicated in the included studies.^cMeans and upper percentiles = soil or soil and dust combined.^dAcid-insoluble residue.^eDust-soil combined.^fBest tracer method.

39	82	246	Davis et al. (1990)					
65 ^e	160 ^c	268	Calabrese et al. (1989)					
153	154	218	85	223	276	1,432	106	Calabrese et al. (1989)
154 ^e	483 ^c	170 ^e	65 ^e	478 ^e	653 ^c	1,059 ^e	159 ^e	Calabrese et al. (1989)
122	139	271	165	254	224	279	114	Stanek and Calabrese (1995a)
133 ^f				217 ^f				Stanek and Calabrese (1995a)
69–120								Van Wijnen et al. (1990)
66 ^f				280 ^f				Calabrese et al. (1997)
196 ^c				994 ^c				Calabrese et al. (1997)

^aAdapted from U.S. EPA (2008) and data contained in the indicated studies.

^b“Upper percentile” as indicated in the included studies.

^cMeans and upper percentiles = soil or soil and dust combined.

^dAcid-insoluble residue.

^eDust+soil combined.

^fBest tracer method.

TABLE 7.18 Estimated Daily Intakes of Dust (mg/day) by Young Children^{a–c}

Methods	Median (50th Percentile)	90th Percentile	95th Percentile	Mean	Maximum
Median of best 4	–6	209	353	17	684
Best tracer	27	559	614	127	1,499
2nd best tracer	8	356	410	83	1,685

^aData from Calabrese et al. (1997).

^bUsing “Best Trace Elements” method for 64 children in Anaconda, MT.

^cMedian of best 4, best tracer, and 2nd best tracer.

quantities, especially interior dusts, that this pathway is considered a major one for child Pb exposure (Lanphear et al., 1998). Calabrese et al. (1997) estimated daily dust ingestion rates in young children using a combination of tracers they noted as “Best Tracer Method.” Results are presented in Table 7.18. The median daily dust ingestion rate using “best tracer” was 27 mg/day, with the corresponding mean, maximum, 90th and 95th percentile values being 127, 1,499, 559, and 644 mg/day, respectively. Overall, it appears that amounts of dust ingested daily by children rival quantities of soil ingested.

REFERENCES

- Adams, M.A., 1991. FDA total diet study: dietary intakes of lead and other chemicals. *Chem. Speciation Bioavailability* 3, 37–41.
- Arcus-Arth, A., Blaisdell, R.J., 2007. Statistical distributions of daily breathing rates for narrow age groups of infants and children. *Risk Anal.* 27, 97–110.
- Binder, S., Sokal, D., Maughan, D., 1986. Estimating soil ingestion: the use of tracer elements in estimating the amount of soil ingested by young children. *Arch. Environ. Health* 41, 341–345.
- Bolger, P.M., Carrington, C.D., Capar, S.G., Adams, M.A., 1991. Reductions in dietary lead exposure in the United States. *Chem. Speciation Bioavailability* 3, 31–36.
- Calabrese, E.J., Stanek III, E.J., 1995. Resolving intertracer inconsistencies in soil ingestion estimation. *Environ. Health Perspect.* 103, 454–457.
- Calabrese, E.J., Barnes, R., Stanek III, E.J., Pastides, H., Gilbert, C.E., Veneman, P., et al., 1989. How much soil do children ingest: an epidemiological study. *Regul. Toxicol. Pharmacol.* 10, 123–137.
- Calabrese, E.J., Stanek, E.J., Gilbert, C.E., 1991. Evidence of soil–pica behavior and quantification of soil ingested. *Hum. Exp. Toxicol.* 10, 245–249.

- Calabrese, E.J., Stanek III, E.J., Pekow, P., Barnes, R., 1997. Soil ingestion estimates for children residing on a Superfund site. *Ecotoxicol. Environ. Saf.* 36, 258–268.
- Clausing, P., Brunekreef, B., van Wijnen, J.H., 1987. A method for estimating soil ingestion by children. *Int. Arch. Occup. Environ. Health* 59, 73–82.
- Davis, S., Waller, P., Buschbom, R., Ballou, J., White, P., 1990. Quantitative estimates of soil ingestion in normal children between the ages of 2 and 7 years: population-based estimates using aluminum, silicon, and titanium as soil tracer elements. *Arch. Environ. Health* 45, 112–122.
- Egan, S.K., Tao, S.S.-H., Pennington, J.A.T., Bolger, P.M., 2002. U.S. Food and Drug Administration's Total Diet Study: intake of nutritional and toxic elements, 1991–1996. *Food Addit. Contam.* 19, 103–125.
- Food and Agriculture Organization: World Health Organization, 1993. Evaluation of certain food additives and contaminants. Forty-First Report of the Joint FAO Expert Committee on Food Additives. Technical Report Series 837. Geneva, Switzerland.
- Galal-Gorchev, H., 1991. Global overview of dietary lead exposure. *Chem. Speciation Bioavailability* 3, 5–11.
- Gulson, B.L., Mizon, K.J., Korsch, M.J., Palmer, J.M., Donnelly, J.B., 2003. Mobilization of lead from human bone tissue during pregnancy and lactation—a summary of long-term research. *Sci. Total Environ.* 303, 79–104.
- Lanphear, B.P., Matte, T.D., Rogers, J., Clickner, R.P., Dietz, B., Bornschein, R.L., et al., 1998. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels: a pooled analysis of 12 epidemiological studies. *Environ. Res.* 79, 51–68.
- Layton, D.W., 1993. Metabolically consistent breathing rates for use in dose assessments. *Health Phys.* 64, 23–36.
- Pennington, J.A.T., 1983. Revision of the total diet study food list and diets. *J. Am. Diet. Assoc.* 82, 166–173.
- Stanek III, E.J., Calabrese, E.J., 1995a. Daily estimates of soil ingestion in children. *Environ. Health Perspect.* 103, 276–285.
- Stanek III, E.J., Calabrese, E.J., 1995b. Soil ingestion estimates for use in site evaluations based on the best tracer method. *Hum. Ecol. Risk Assess.* 1, 133–156.
- U.S. Department of Agriculture: Agricultural Research Service, 1990. Continuing Survey of Food Intakes by Individuals 1987–1988. Food Surveys Research Group, Beltsville, MD.
- U.S. Department of Agriculture: Agricultural Research Service, 1998. Continuing Survey of Food Intakes by Individuals 1994–96, 1998. Food Surveys Research Group, Beltsville, MD.
- U.S. Environmental Protection Agency, 1986. Air Quality Criteria for Lead. Report No. EPA/600/8-83/028bF. Environmental Criteria and Assessment Office, Washington, DC (Chapter 10).
- U.S. Environmental Protection Agency, 1994. Guidance Manual for the Integrated Exposure-Uptake Biokinetic Model for Lead in Children. Report No. EPA/540-R93/081. Office of Research and Development, Washington, DC.
- U.S. Environmental Protection Agency, 1995. National Air Quality and Emissions Trends Report, 1994. Report No. EPA 454/R-95-014. Office of Air and Radiation, Washington, DC.
- U.S. Environmental Protection Agency, 2006. Air Quality Criteria for Lead. Vol. 1. Report No. EPA/600/R-05/144aF. National Center for Environmental Assessment, Washington, DC.
- U.S. Environmental Protection Agency, 2008. Child-Specific Exposure Factors Handbook. Report No. EPA/600/R-06/096F. National Center for Environmental Assessment, Washington, DC.
- U.S. Environmental Protection Agency, 2009. IEUBK win32 Lead Model Version 1.1. Build 11. <<http://www.epa.gov/superfund/health/contaminants/lead/index.htm>> (accessed 09.03.11.).

- ow, P., Barnes, R., 1997. Soil ingestion estimates for chil-
Ecotoxicol. Environ. Saf. 36, 258-268.
- Wijnen, J.H., 1987. A method for estimating soil ingestion by
on. Health 59, 73-82.
- Wijnen, J., White, P., 1990. Quantitative estimates of soil inges-
the ages of 2 and 7 years: population-based estimates using ab-
oil tracer elements. Arch. Environ. Health 45, 112-122.
- Wijnen, J.A.T., Bolger, P.M., 2002. U.S. Food and Drug
Study: intake of nutritional and toxic elements, 1991-1996
25.
- World Health Organization, 1993. Evaluation of certain
s. Forty-First Report of the Joint FAO Expert Committee on
ort Series 837. Geneva, Switzerland.
- overview of dietary lead exposure. Chem. Speciation
- M.J., Palmer, J.M., Donnelly, J.B., 2003. Mobilization of
during pregnancy and lactation—a summary of long-term
03, 79-104.
- s, J., Clickner, R.P., Dietz, B., Bornschein, R.L., et al., 1998.
aminated house dust and residential soil to children's blood
of 12 epidemiological studies. Environ. Res. 79, 51-68.
- consistent breathing rates for use in dose assessments. Health
- of the total diet study food list and diets. J. Am. Diet. Assoc.
- 1995a. Daily estimates of soil ingestion in children. Environ.
5.
- 1995b. Soil ingestion estimates for use in site evaluations based
m. Ecol. Risk Assess. 1, 133-156.
- Agricultural Research Service, 1990. Continuing Survey of
1987-1988. Food Surveys Research Group, Beltsville, MD.
- Agricultural Research Service, 1998. Continuing Survey of
1994-96, 1998. Food Surveys Research Group, Beltsville, MD.
- Agency, 1986. Air Quality Criteria for Lead. Report No. EPA/
ental Criteria and Assessment Office, Washington, DC
- Agency, 1994. Guidance Manual for the Integrated Exposure-
or Lead in Children. Report No. EPA/540-R93/081. Office of
Washington, DC.
- Agency, 1995. National Air Quality and Emissions Trends
PA 454/R-95-014. Office of Air and Radiation, Washington, DC.
- Agency, 2006. Air Quality Criteria for Lead. Vol. 1. Report No.
onal Center for Environmental Assessment, Washington, DC.
- Agency, 2008. Child-Specific Exposure Factors Handbook
06/096F. National Center for Environmental Assessment,
- Agency, 2009. IEUBK win32 Lead Model Version 1.1. Build 11.
fund/health/contaminants/lead/index.htm> (accessed 09.03.11).
- U.S. Food and Drug Administration, 2004. U.S. FDA Total Diet Study—analytical results.
<<http://www.fda.gov/Food/FoodSafety/FoodContaminantsAdulteration/TotalDietStudy/ucm184293.htm>> (accessed 03.11.10.).
- Wijnen, J.H., Clausen, P., Brunekreef, B., 1990. Estimated soil ingestion by children.
Environ. Res. 51, 147-162.